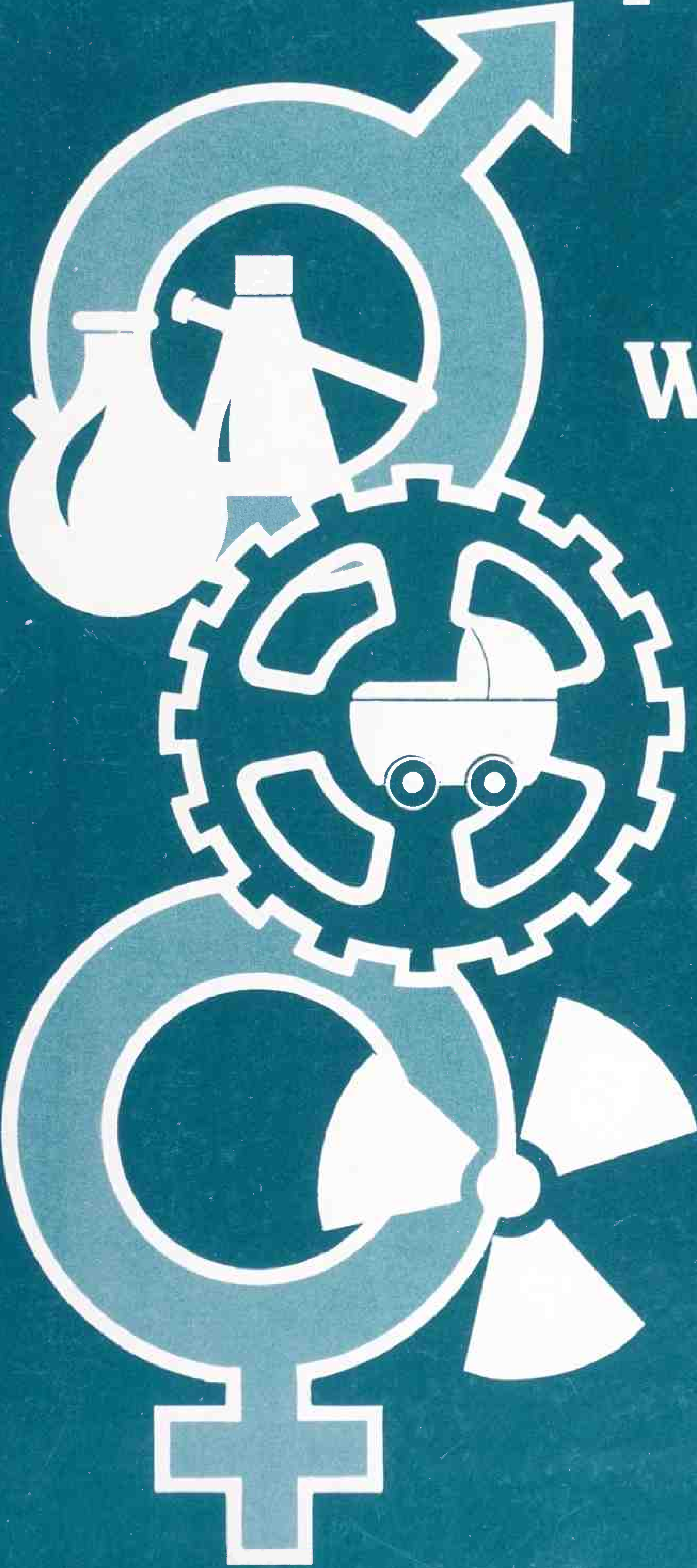
Reproductive Health Hazards in the Workplace



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Reproductive Health Hazards in the Workplace

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Foreword

This report reviews current knowledge of hazards and suspected hazards to the reproductive health of America's working men and women and to the health and well-being of their children. The analysis was requested by the House Committee on Science and Technology, with letters of support from the Senate Committee on Labor and Human Resources, and the House Committee on Agriculture, Subcommittee on Department Operations, Research, and Foreign Agriculture.

The nature and actions of the chemical, biological, and physical factors that may cause reproductive or developmental impairment are described, as is the complexity of measuring reproductive endpoints. The first section reviews current technologies for assessing reproductive function, and examines the human and animal studies conducted to determine the extent of risk posed by suspected agents and the difficulties in interpreting study findings for this purpose.

The report then reviews the role of the regulatory process in preventing workplace exposure to reproductive health hazards, and the legal redress from either State workers' compensation systems or the tort system that is available to those affected. This section also analyzes sex discrimination issues arising from the fact that protection policies instituted in hospitals and industry have, in certain instances, discriminated against women workers. The third section discusses the ethical principles underlying the protection of reproductive health in the workplace.

The Office of Technology Assessment was assisted in the preparation of this study by an advisory panel of individuals selected to reflect both the substantive issues and the relevant social issues covered in the assessment. Panelists were drawn from academia, industry, trade associations, public interest groups, and labor unions. Their areas of scientific expertise included reproductive and developmental toxicology, male and female reproductive biology, and epidemiology. Legal interests included sex discrimination, workers' compensation, tort, and regulatory law. Eighty-nine reviewers drawn from universities, trade associations, the executive branch, and the private sector provided helpful comments on draft reports.

The Office expresses sincere appreciation to each of these individuals. As is the case with all OTA reports, however, the content of this report is the responsibility of the Office and does not necessarily constitute the consensus or endorsement of the advisory panel or the Technology Assessment Board.

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CONTENTS

Page

Glossary of Acronyms and Terms ix

Chapter

1. Executive Summary 3
2. Introduction to the Study 31
3. Principles of Reproductive Biology and Development 43
4. Evidence for Workplace Hazards to Reproductive Function 67
5. Technologies for Assessing Human Reproductive Function 129
6. Reproductive Risk Assessment 161
7. The Regulatory Process 181
8. Sex Discrimination Issues 235
9. Workers' Compensation 279
10. Tort Liability for Reproductive Harm 301
11. The Ethical Issues 329

Appendix *Page*

A. Reproductive Dysfunction in the Population 341
B. Sample Patient History Questionnaire 365
C. Technical Notes: OSHA 390
D. Technical Notes: EPA 400
E. NRC Regulation of Exposure 405
F. List of Contractor Reports, Working Papers, and Staff Papers 407
G. List of Contributors and Acknowledgments 408

Index 413

Glossary of Acronyms and Terms

Acronyms

AEC —Atomic Energy Commission

ACGIH —American Conference of Governmental Industrial Hygienists

ALARA —as-low-as-reasonably achievable (see *Terms*)

ANSI —American National Standards Institute

BATF —Bureau of Alcohol, Tobacco and Firearms

BDMP —Birth Defects Monitoring Program

BFOQ —bona fide occupational qualification

BNA —Bureau of National Affairs (publisher)

BLS —Bureau of Labor Statistics (DOL)

CDC —Centers for Disease Control (PHS, DHHS)

CNS —central nervous system

C.F.R. —Code of Federal Regulations

CEQ —Council on Environmental Quality

CPSC —Consumer Product Safety Commission

CSIN —Chemical Substances Information Network

DBCP —dibromochloropropane

DDT —2,2-bis[p-chloro-phenyl]1,1,1,-trichloroethane)

DES —diethylstilbestrol

DHHS —Department of Health and Human Services

DNA —deoxyribonucleic acid

DOL —Department of Labor

EDB —ethylene dibromide

EEOC —Equal Employment Opportunity Commission

EPA —U.S. Environmental Protection Agency

EtO —ethylene oxide

ETS —Emergency Temporary Standard

FACOSH —Federal Advisory Council on Occupational Safety and Health

FDA —Food and Drug Administration

FIFRA —Federal Insecticide, Fungicide, and Rodenticide Act

FPP —fetal protection policy

FRC —Federal Radiation Council

FSH —follicle-stimulating hormone

FTCA —Federal Tort Claims Act

HANES —National Health and Nutrition Survey (NCHS)

hCG —human chorionic gonadotropin

ICRP —International Commission on Radiation Protection

ITC —Interagency Testing Committee (EPA)

ITSDC —Interagency Toxic Substance Data Committee (EPA)

LH —luteinizing hormone

LHRH —luteinizing hormone-releasing hormone

MSDS —Material Safety Data Sheet

MSH Act —Mine Safety and Health Act

MRP —Medical Removal Protection

NACOSH —National Advisory Committee on Occupational Safety and Health

NAS —National Academy of Sciences

NSF —National Science Foundation

NCRP —National Council on Radiation Protection and Measurements

NIEHS —National Institute of Environmental Health Sciences (NIH)

NIH —National Institutes of Health

NIOSH —National Institute for Occupational Safety and Health (CDC)

NOEL —no observed effect level

NTP —National Toxicology Program

NRC —Nuclear Regulatory Commission

OERC —Occupational Exposure Review Committee

OFCCP —Office of Federal Contract Compliance Programs

OMB —Office of Management and Budget

OSH Act —Occupational Safety and Health Act

OSHA —Occupational Safety and Health Administration (DOL)

OSHRC —Occupational Safety and Health Review Commission

PBB —polybrominated biphenyls

PCB —polychlorinated biphenyls

PDA —Pregnancy Discrimination Act

PEL —permissible exposure limits

PHS —Public Health Service

PMA —Petition for Modification of Abatement

PMN —premanufacture notification

PVC —polyvinyl chloride

REAG —Reproductive Effects Assessment Group

RPAR —Rebuttable Presumption Against Registration

RR —rate retention

SIC —Standard Industrial Classification

SNUR —Significant New Use Rule (EPA)

STEL —Short-term Exposure Limit
TCDD —2,3,7,8-tetrachlorodibenzo-p-dioxin
TCP —2,4,5 trichlorophenol
TLV —threshold limit value
TSCA —Toxic Substances Control Act
USDA —U.S. Department of Agriculture
VDT —video display terminal

Terms

Administrative controls: Methods of reducing worker exposures to occupational hazards through management arrangements; e.g., rotating workers from high- to low-exposure areas to reduce average exposure level, scheduling jobs or processes that generate hazards during times when few workers are present.

Agent Orange: A 50/50 mixture of 2,4,-D and 2,4,5-T widely used as a defoliant during the Vietnam war.

ALARA (assumption): "As-low-as-reasonably achievable." A public health principle which holds that exposures to hazards be kept at or below levels permitted by established standards.

Amenorrhea: The absence or abnormal cessation of menstruation; normal before puberty, after the menopause, during pregnancy and lactation.

Amniocentesis: The extraction of amniotic fluid for diagnostic purposes.

Anencephaly: A congenital deformity in which the brain is absent.

Apgar score: Numerical expression of an infant's condition 60 seconds after birth, based on heart rate, respiration, muscle tone, color, response to stimuli.

Azoospermia: The complete absence of sperm.

Basal body temperature: Body temperature during rest or inactivity; commonly obtained upon awakening.

Beneficence: Moral principle that requires avoiding harms to others and maximizing the balance of benefits over harms.

Blastocyst: See *embryo/fetus*.

BFOQ exception: An exception to Title VII's prohibition against employment policies that intentionally discriminate against one sex. Intentional discrimination is permitted if sex is a bona fide occupational qualification (e.g., a male actor to portray a male character).

Birth defect: Any structural, functional, or biochemical abnormality, whether genetically determined or induced during gestation, that is not due to injuries suffered during birth.

Breach of warranty: As used in the common law of contracts, breach of warranty is the failure or falsehood of an affirmative promise or statement. Unlike fraud, it does not involve guilty knowledge. Under the Uniform Commercial Code, a violation of either an express or implied warranty for which an action in contract will lie. U.S.C. § 2-312 et seq.

Business necessity exception: An exception to Title VII's prohibition against sex-neutral employment policies that have a disparate impact on one sex. A policy with a disparate impact on one sex is permissible if the policy is necessary to achieve a business purpose. Similar to the BFOQ exception, but used in cases where discriminatory effect rather than discriminatory intent is at issue. (See also *disparate impact*.)

Carbaryl: 1-Napthyl methyl carbamate, a broad-spectrum insecticide.

Causation: The act by which an effect is produced. An important doctrine in fields of negligence and product liability law.

Carcinogen/carcinogenesis: A substance or physical agent that causes cancer.

Childbearing years: The reproductive age span of women, assumed for statistical purposes to be 15 to 44.

Chlordecone: See *Kepone*.

Chorionic villus biopsy: A prenatal diagnostic technique that permits early identification of various disorders, particularly genetically based diseases.

Confounding factor: A variable that is related to both the exposure and the outcome being studied.

Congenital: Present at birth.

Corpus luteum: Remnant of ovulated follicle within ovary; secretes progesterone.

DDT (2,2-bis (p-chloro-phenyl) 1,1,1,-trichloroethane): A pesticide in common use around the world that mimics the effects of estrogen. U.S. use was halted in 1972.

Developmental abnormality: Structural or functional defect occurring during gestation.

Developmental toxin/toxicity: An agent that impedes proper anatomical or physiological development of offspring. May act at any point between conception and puberty.

Dibromochloropropane (DBCP): A chemical used as a pesticide. Most uses of DBCP are now prohibited by law.

Dioxin: 2,3,7,8,-tetrachlorodibenzo-p-dioxin, or TCDD. An unwanted contaminant of the synthesis leading to 2,4,5,-T and other chemicals.

Discriminatory effects, discriminatory impact: See *disparate impact*.

Discriminatory treatment: See *facial discrimination*.

Disparate impact: Used to describe employment policies that are not intended to be discriminatory but nevertheless are disproportionally burdensome on members of one sex. Such policies violate Title VII unless considered a "business necessity."

Dominant lethal: A gene, either a new mutation or inherited from one parent, that causes death of the organism.

Dose-response assessment: In the risk assessment process, determines the relationship between the magnitude of human exposure and the probability of human health effects.

Dual capacity exception: Exception to the exclusivity of remedy doctrine in the workers' compensation laws of some States that permits employee suits against employers for personal injuries if the employer can be viewed as causing the injury in a capacity other than employment (e.g., if the employer also acted as a product manufacturer or provider of medical services). (See also *exclusivity of remedy doctrine.)*

EDB: See *ethylene dibromide.*

Embryo/fetus: The embryonic stage begins at about 3 weeks and extends to about 8 or 9 weeks; the fetal stage extends from 8 weeks until birth. The first or blastocyst stage is often subsumed within the embryonic stage to simplify terminology.

Embryotoxin/embryotoxicity: A agent that adversely affects the embryo. (See *toxin/toxicity.)*

Emergency temporary standard (ETS): A standard issued under § 6(c) of the Occupational Safety and Health Act which may be issued when OSHA determines that workers are exposed to a "grave danger" from an occupational hazard and that an emergency standard is necessary to protect them from that danger.

Endometrium: The mucous membrane of the uterus, which varies in thickness and structure with each phase of the menstrual cycle.

Endpoint: The particular biological response being measured.

Engineering controls: Methods of controlling worker exposure by modifying the source or reducing the amount of contaminants released into the workplace. Engineering controls include process design and modification, equipment design, enclosure and isolation, and ventilation.

Epidemiology: The study of the distribution of diseases and their precursors in human populations.

Estrogen: Any natural or artificial substance that induces estrogenic activity; more specifically the estrogenic hormones estradiol and estrone produced by the ovary; the female sex hormones.

Ethylene dibromide (EDB): A chemical used chiefly as a gasoline additive and as a pesticide from 1948 to 1984, when it was banned for pesticidal use.

Ethylene oxide (EtO): A clear, colorless gas used primarily as a chemical intermediate in the production of pesticides and as a sterilant and fumigant for hospital equipment.

Etiology: The study of the causes of disease.

Exclusivity of remedy doctrine: A provision of all State workers' compensation laws that prohibits employee tort suits against employers for injuries or diseases that occur on the job.

Exclusivity rule: See *exclusivity of remedy doctrine.*

Exposure: The length of time and dose of chemical, biological, or physical agent to which a worker is subjected.

Exposure assessment: In risk assessment, identifies the population segments potentially exposed to the agent.

Facial discrimination: Employment discrimination of an overt and intentional nature, such that the employment policy is considered to be discriminatory on its face. Facial sex discrimination violates Title VII unless sex is a "bona fide occupational qualification." Compare with *disparate impact.*

Fetal protection policy (FFP): An occupational health policy intended to provide for the protection of the future offspring of employees. Such policies frequently place limitations on the jobs available to fertile and/or pregnant women.

Fetoscopy: A procedure using an optical instrument that allows direct observation of the fetus.

Fetotoxin/fetotoxicity: An agent that adversely affects the fetus. (See *toxin/toxicity.*)

Fetus: See *embryo/fetus.*

Follicle-stimulating hormone (FSH): A protein secreted by the anterior pituitary that promotes spermatogenesis and stimulates ovulation.

Gamete: A mature male or female germ cell (spermatozoon or ovum).

General duty clause: Section 5(a)(1) of the Occupational Safety and Health Act. This section provides that "each employer shall furnish . . . employment and a place of employment which are free from recognized hazards that are causing or are likely to cause death or serious physical harm to his employees." OSHA has used this clause to cite employers for workplace conditions that present serious occupational hazards that are not covered by OSHA's more detailed health and safety standards.

Genome: The total genetic information carried by an individual.

Germ cell: The male and female reproductive cells; egg and sperm.

Gestation: Period of intrauterine development from conception to birth.

Gonad: A generic term that refers to both female ovaries and male testes.

Gonadotropin: A substance having affinity for or a stimulating effect on the gonads. There are three varieties: anterior pituitary, chorionic from human pregnancy urine, and chorionic from the serum of pregnant mares.

Hazard identification: In risk assessment, the qualitative analysis of all available experimental animal and human data to determine whether and at what dose an agent is likely to cause toxic effects.

Human chorionic gonadotropin (hCG): A hormone produced by the placenta that stimulates production of progesterone.

Hydrocephaly: Abnormal accumulation of fluid in the cranium, associated with mental retardation.

Hysterosalpingogram: Imaging of the uterus and fallopian tubes by injecting dye into the cervix and filming its spread through the peritoneal cavity.

Iatrogenic: Treatment- or drug-induced.

Implantation: Process whereby a fertilized ovum burrows into the lining of the uterus on its arrival there, and attaches itself firmly. Successful implantation is essential to the future development of the embryo/fetus and is sometimes considered as the true moment of conception.

Infant mortality rate: The number of deaths to infants under 1 year of age in a given year per 1,000 live births in that year.

Infertility: Inability to produce liveborn children.

Intentional tort exception: Exception to the exclusivity of remedy doctrine in some States that permits an employee lawsuit against an employer if the employer's conduct manifested a deliberate attempt to injure the worker.

In vitro: Outside the living organism and in an artificial environment.

In vivo: Within the living organism.

Ionizing radiation: Energy that is transmitted in wave or particle form that is capable of causing ionization (ejecting orbital electrons) of atoms or molecules in radiated tissue; e.g., X-rays.

Job-relatedness (causation): A criterion for receiving worker's compensation benefits that requires the worker's disability, injury, or disease be caused by a workplace factor.

Justice: As a principle of ethics, fair and equal treatment of others.

Karyotyping: A technique by which chromosomes are prepared for microscopic observation; a standard part of amniocentesis.

Kepone (chlordecone): A chlorinated hydrocarbon insecticide, used commonly against fire ants and cockroaches; U.S. use was banned in 1977.

Laparoscopic ovarian biopsy: Use of a laparoscope to remove a portion of ovarian tissue for microscopic observation.

Laparoscope: An instrument used for direct observation of ovaries and other internal organs.

Loss of consortium: Loss of the conjugal fellowship of husband or wife, and the right of each to the company, society, cooperation, affection, and aid of the other in every conjugal relation. Damages for loss of consortium are commonly sought in wrongful death actions, or when spouse has been seriously injured through negligence of another, or by spouse against third person alleging that he or she has caused breaking up of marriage.

Luteal phase: The portion of the menstrual cycle that occurs between ovulation and menses.

Luteinizing hormone (LH): A pituitary hormone that stimulates hormone production by gonads.

Luteinizing hormone-releasing hormone (LHRH): A hormone released by the hypothalamus that regulates reproductive function in men and women.

Medical removal protection (MRP): An employment policy requiring or permitting employees to transfer permanently or temporarily from jobs involving a potential health risk to jobs with less risk.

Menarche: The beginning of menstruation; i.e., the first menstrual period. This occurs during puberty but does not signify the beginning of full adult fecundity as ovulation may be irregular or absent for some time.

Menopause: Natural physiologic cessation of menstruation normally occurring in the last half of the fifth decade.

Microcephaly: Abnormal smallness of the head.

Morbidity: The frequency of disease and illness in a population.

Mutagen/mutagenesis: A substance that induces mutation; the induction of mutation in the genetic material.

Neonate: A newborn infant.

Neural tube defects: Birth defects of the central nervous system such as spina bifida and anencephaly.

Nonionizing radiation: Refers to the region of the electromagnetic spectrum where the energy of the emitted photon is incapable of ionizing atoms or molecules in the irradiated tissue; e.g., radio and television transmission signals.

No observed effect level (NOEL): Level of exposure that produces no observed deleterious health effects.

Oligospermia: Extremely low levels of sperm production.

Oocyte: Female germ cell.

Organogenesis: The formation and development of body organs from embryonic tissues.

Ovulation: The release of an ovum from the ovary during the female menstrual cycle.

Parturition: Labor, giving birth.

Parity: The number of pregnancies a woman has carried to at least 20 weeks gestation (or 500-gram fetal weight).

Permissible exposure limit (PEL): The maximum airborne concentration of a toxic substance permitted by OSHA standards.

Personal protective equipment: Equipment and clothing designed to control exposure to hazards; e.g., hard hats, safety shoes, protective eyewear, protective clothing and gloves, hearing protectors, and various types of respirators, such as dust and gas masks.

"Personal" injury or disease: A criterion for receiving workers' compensation benefits that prohibits claims by the worker's spouse or offspring. The injury or disease must be "personal" to the worker.

Pharmacokinetics: The study of the action of a chemical in the body over a period of time. It includes the processes of absorption, distribution, localization in tissues, transformation into other chemicals with biological activity, and excretion.

Polybrominated biphenyl (PBB): A chemical used as a flame retardant in thermoplastic products until banned in 1979.

Polychlorinated biphenyl (PCB): A chemical used in coolant fluid in electrical transformers, hydraulic fluids, lubricants, and as a pesticide extender until banned in 1979.

ppm: Parts per million.

Pre-conception tort: A wrongful act committed prior to the conception of the offspring injured as a consequence of the act.

Premanufacture notification (PMN): Requirement under TSCA that companies must notify EPA before commencing manufacture of toxic substances.

Prenatal tort: A wrongful act committed after conception but prior to the birth of the offspring injured as a consequence of the act.

Preponderance of evidence: Evidence that is of greater weight or more convincing than the evidence that is offered in opposition to it, that is, evidence which as a whole shows that the fact sought to be proved is more probable than not. With respect to burden of proof in civil actions, means greater weight of evidence, or evidence that is more credible and convincing to the mind.

Product liability theory: The legal liability of manufacturers and sellers to compensate buyers, users, and even bystanders for damages and injuries suffered because of defects in the goods purchased. A tort which makes a manufacturer liable if his product has a defective condition that makes it unreasonably dangerous to the user or consumer.

Progesterone: A steroid hormone obtained from the corpus luteum, adrenals, or placenta. It is responsible for changes in uterine endometrium in the second half of the menstrual cycle that prepare for implantation of the blastocyst, development of maternal placenta after implantation, and development of mammary glands.

Rads: The units used to quantify the energy deposited in matter by ionizing radiation, defined as 0.01 joules per kilogram of irradiated material.

Rate retention (RR): Maintaining the removed employee's wages and benefits during the period of medical removal. (See also *medical removal protection*.

Rational basis test: The legal test applied by a court that is reviewing the constitutionality of a decision of a legislative or administrative body. A court will not second-guess the legislature as to the wisdom or rationality of a particular statute if there is a rational basis for its enactment when the strict scrutiny test does not apply.

Reasonable personal standard: The standard that one must observe to avoid liability or negligence is the standard of the reasonable person under all the circumstances, including the foreseeability of harm to one such as the plaintiff.

Rem: Abbreviation for roentgen equivalent measure, a unit that quantifies the degree of biological damage from ionizing radiation.

Reproductive age: See *childbearing years*.

Reproductive health hazard: A chemical, physical, or biological agent that causes reproductive impairment in adults and developmental impairment or death in the embryo/fetus or child.

Reproductive toxin: An agent that interferes with reproductive or procreative functioning of the adult from puberty through adulthood.

Respect for persons: A moral principle that requires that individuals be treated as the focus of concern in their own right and not merely as the means to the achievement of other goals.

Risk assessment: The use of scientific evidence to estimate the likelihood of adverse effects on the health of individuals or populations from exposure to hazardous materials and conditions.

Risk characterization: In risk assessment, the final step, which summarizes information about the agent and evaluates it in order to estimate the risk.

Risk management: Determination of the possible actions that can or should be taken in response to an assessment that a substance or condition poses a significant risk.

Semen: A mixture of sperm and fluids.

Sex ratio: The ratio of males to females in a population, usually expressed as the number of males for every 100 females.

Somatic cell: All cells of the body except the germ cells.

Sonographic imaging: See *ultrasonography*.

Sovereign immunity: Doctrine that precludes a litigant from asserting an otherwise meritorious cause of action against a sovereign (government) or a party with sovereign attributes unless sovereign consents to suit. Historically, the Federal and State governments, and derivatively cities and towns, were immune from tort liability arising from activities that were governmental in nature. More jurisdictions, however, have abandoned this doctrine in favor of permitting tort actions with certain limitations and restrictions.

Spermatogenesis: The transformations that result in formation of spermatozoa.

Spermatogonia: Precursor sperm cells.

Spermatozoa: Sperm cell.

Spina bifida: A neural tube defect characterized by incomplete closure of the spinal column.

Steroid hormones: See *estrogen*, *progesterone*.

Survival statute: Statutory provision for the survival, after death of the injured person, of certain causes of action for injury to the person, whether death results from the injury or from some other cause.

TCDD: See *dioxin*.

Threshold limit value (TLV): Maximum airborne concentrations of toxic substances set as guidelines by the ACGIH.

Teratogen/teratogenesis: An agent that interferes with embryonic or fetal development. A chemical or physical agent that causes physical defects in offspring.

Testosterone: The hormone secreted by the testes that stimulates the development of masculine characteristics.

Tort: A wrongful act for which the law imposes liability.

Toxicant: See *toxin*.

Toxin/toxicity: A chemical, physical, or biological agent that interrupts the normal function of a cell, tissue, organ, or organism.

2,4,-D: 2,4-dichlorophenoxyacetic acid; an herbicide commonly used in agriculture and forestry.

2,4,5,-T: 2,4,5,-trichlorophenoxyacetic acid, a chlorinated herbicide in wide U.S. use from 1948 to 1970. Banned in 1979 for all use except on rangeland and rice fields.

Ultrasonography: Imaging of the ovaries or developing embryo/fetus using sonic waves.

Viability: A concept used to distinguish between the early stages of gestation, when the embryo or fetus is incapable of survival outside the uterus, and the later stages, when the fetus can live outside the womb. Given current neonatal technologies, a fetus achieves viability after approximately 6 months of gestation.

Workers' compensation: State-required insurance programs that pay for an employee's medical costs and other economic costs due to work-related injury and illness.

Wrongful death: A death resulting from a tort. Some States have enacted special statutes, known as wrongful death acts, to address liability in such cases. These statutes generally do not apply to fetal deaths.

Wrongful birth, wrongful life: A life resulting from a tort, usually the birth of an infant with birth defects as a result of a health care provider's negligent failure to either inform the parents of the risk of birth defects or to perform procedures with due care to prevent conception or birth. Wrongful birth refers to the parent's claim for damages, while wrongful life refers to the child's claim for damages. The courts are divided as to whether relief can be granted for such claims.

Zygote: Fertilized egg; the result of the union of sperm and ovum.

Chapter 1

Executive Summary

CONTENTS

Page

Introduction 3

Reproductive Biology and Mechanisms of Toxic Effects 5

Evidence for Workplace Hazards to Reproductive Function 6

Reproductive Risk Assessment 7

Reproductive Risk Assessment in the Regulatory Process 9
  Occupational Safety and Health Administration 9
  Environmental Protection Agency 10
  Nuclear Regulatory Commission 11

Sex Discrimination 11

Workers' Compensation 12

Tort Liability 13

Ethical Considerations 14
  Respect for Persons 14
  Beneficence 15
  Justice 15

Issues and Options 16
  Sex Discrimination 16
  Regulation 21
  Compensation for Job-Induced Reproductive Harm 24
  Reducing Uncertainty: Issues in Research 26

List of Tables

Table No. *Page*

1-1. Agents and Substances Reviewed for Reproductive Health Effects by OTA 7

1-2. Summary of Harms, Victims, Benefits Criteria, and Causation Problems in Workers' Compensation Systems 13

Chapter 1

Executive Summary

INTRODUCTION

Protecting the reproductive health and procreative capacity of working men and women is important for two basic reasons: 1) it safeguards the health of future generations, and 2) reproductive health and procreative capacity are fundamentally important to individual well-being.

Reproductive health hazards, for the purpose of this report, are defined as agents that cause reproductive impairment in adults and developmental impairment or death in the embryo/fetus[1] or child. The effects of reproductive impairment, which can include infertility, impotence, menstrual irregularities, spontaneous abortion, and damage to offspring, are difficult to measure and can result in damage to other, related systems of the body. Individuals also vary widely in susceptibility and extent of exposure to reproductive hazards.

What is known about reproductive health hazards is far outweighed by what is unknown: most commercial chemicals have not been thoroughly evaluated for their possible toxic effects on reproduction and development. Much of the information on suspected reproductive health hazards, as with other hazards, is derived from animal studies, which present problems of interpretation in extrapolating to effects in humans.

There are consequently no reliable estimates as yet of the basic measures of reproductive risk in the workplace—the number of workers exposed to such hazards, their levels of exposure, and the toxicity of the agents to which they are exposed.

There are a number of sophisticated technologies for assessing reproductive function, but none can fully assess fertility; the only true measure is the birth of a healthy infant. **Because of these unknowns, the management of uncertainty is the central issue in the protection of the reproductive health and procreative capacity of working men and women.**

Most policy decisions regarding the management of occupationally related reproductive risk must be made within the context of two Federal statutes:

1. the Occupational Safety and Health Act (OSH Act), which gives the Federal Government the authority to protect workers to the extent feasible from exposure to substances that could damage their reproductive systems and general health; and
2. Title VII of the Civil Rights Act, which forbids employment discrimination on the basis of sex or pregnancy.

The OSH Act and the Civil Rights Act can usually be reconciled in cases where protection of the health of the embryo/fetus is of concern. An employer who employs in a nondiscriminatory manner and provides a place of employment that is free of recognized hazards violates neither law. When there is risk of exposure to recognized hazards in the workplace, the employer is obliged to take all reasonable nondiscriminatory steps to ameliorate the hazard. Employers who are nevertheless unable to provide a safe workplace to all employees may be legally permitted to resort to sex-based distinctions in removing individuals at risk if the employer meets certain stringent criteria established by the courts.

Three additional major statutes potentially apply to occupational reproductive risk—these are the Toxic Substances Control Act (TSCA); the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA); and the Atomic Energy Act (AEA).

A number of hazardous agents have been associated in varying degrees with impairment of male and female reproductive function and the health of the developing embryo/fetus. Their effects are mediated by genetic and environmental factors as well as by exposure.

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[1] Gestation is commonly divided into three stages: 1) the blastocyst, from conception until about 3 weeks; 2) the embryonic, from 3 weeks to about 8 or 9 weeks; and 3) the fetal, from 8 or 9 weeks until birth. The blastocyst stage is often subsumed within the embryonic stage in order to simplify terminology.

These agents include various chemicals; ionizing and nonionizing radiation; physical factors such as hot, cold, hyperbaric, or hypobaric environments, noise, and vibration; infectious agents; aspects of lifestyle such as tobacco and alcohol use; ingestion or absorption of certain drugs; and overexertion and stress.

Toxic agents are regulated for a range of health effects which until recently did not often include reproductive effects. However, toxic agents are unlikely to be regulated solely for their effects on reproductive health because toxic agents that affect reproductive health are likely to have other health effects as well. To date, four health hazards—ionizing radiation, lead, ethylene oxide (EtO), and dibromochloropropane (DBCP)—are regulated in part because of their effects on reproductive or procreative capacity.

Workers have two primary concerns related to reproductive health: exposure to substances that can endanger their reproductive health and procreative capacity, and exposure to substances that can endanger the health and development of their offspring. Workers are also concerned about employment opportunities and job security in this context. For example, employment opportunities for women workers may be affected by fetal protection policies instituted by employers who fear future liability for offspring harmed by workplace exposures. Opinions of workers regarding these policies differ, depending on their values and economic circumstances.

While policymakers and employers may never have complete information regarding the full extent of reproductive dysfunction and its causes, they must attempt to provide as safe a workplace as feasible. The primary means of protecting reproductive health in the workplace are adequate engineering and administrative controls to keep exposure at the lowest feasible levels; substitution of safer substances where feasible; and programs to educate workers concerning safe work practices and potential dangers.[2]

The methods used to protect workers' reproductive health must meet minimum standards under the OSH Act and Title VII. Managers and policymakers often have different approaches to meeting minimum standards, depending on their personal philosophies. One view holds that all workers, even the hypersusceptible, must have equal access to job opportunities. In this view, justice cannot be served if employment is denied on the basis of immutable traits, such as sex, age, ethnic status, or genetic susceptibility. The workplace must therefore be made safe enough to protect the health of even the most vulnerable worker. A contrasting view holds that the hypersusceptible worker may be denied equal access to job opportunities in situations where it is neither technically nor economically feasible to protect that worker. In this view, justice is served because the majority of workers have equal access and the employer can remain in business. Difficulties arise because the evidence that exposure to a substance causes harm is rarely conclusive, people cannot agree on the definition of "safe," and the definition and implications of hypersusceptibility can change, depending on the workplace situation. Thus, depending on philosophical viewpoint, justice can be interpreted to mean either equal opportunity for all or the greatest good for the greatest number.

If protective measures fail and workers are harmed, compensation becomes the issue. Under the laws of most States, reproductive impairment probably cannot be compensated within the workers' compensation system; moreover, workers are at present barred from bringing tort claims against their employers. Although lawsuits against third parties such as product suppliers and manufacturers may achieve redress, proving causation is often difficult. And, in some cases, third-party defendants cannot be identified.

Although it is difficult to identify the agents that are hazardous to reproductive health and the numbers of people who may be exposed, reproductive dysfunction is a significant health problem in the United States:

> An estimated 2.4 million (8.4 percent) of U.S. couples in which the wife is of childbearing age are unintentionally infertile. In some cases this

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[2] See OTA's report on *Preventing Illness and Injury in the Workplace*, OTA-H-256 (Washington, DC: U.S. Government Printing Office, April 1985).

inability to bear children appears to correct itself; in other cases the infertility persists.

Some congenital malformation is evident in 3 percent of all live births; an additional 3 percent of infants are found to have malformations by 1 year of age. The causes of congenital malformations are unknown in 60 to 70 percent of cases. (Rates of congenital malformation do not appear to be rising.)

The rates of other manifestations of reproductive and procreative dysfunction (e.g., depressed libido, impotence, contaminated breast milk, early menopause) are unknown.

Although the extent to which workplace exposure to chemical, physical, and biological agents may contribute to impairment of reproductive functioning is not known, the National Institute for Occupational Safety and Health (NIOSH) ranks work-related reproductive impairment as sixth of the 10 leading work-related diseases and injuries. This ranking is based on numbers of workers exposed to known toxicants or substances suspected of being toxic to human reproductive capacity and levels of reproductive dysfunction in the population. Thus there is a clear need to elucidate the specific causes of reproductive dysfunction in order to reduce its overall incidence.

This report reviews the evidence for workplace-induced reproductive impairment. The options describe actions that might be taken to reduce the uncertainty surrounding its prevalence and causes, and to compensate those who may be harmed.

REPRODUCTIVE BIOLOGY AND MECHANISMS OF TOXIC EFFECTS

The complexity of the reproductive process is often masked by a focus on discrete components of procreation, such as the production of sperm or egg cells or development of the embryo/fetus. This narrow focus fails to encompass such aspects of reproductive function as overall adult health, sexual behavior, pregnancy, lactation, child health and development, puberty, and reproductive senescence. Failure to recognize the integral role of each of these components as part of reproductive function leads to an underestimate of the sensitivity of normal reproductive functioning to even minor disruptions.

The processes involved in the production of sperm and egg cells are different. Men produce sperm continuously from puberty throughout life. By contrast, women are born with a finite supply of egg cells which is steadily depleted from puberty through menopause.

Embryo loss is a part of the reproductive process. Only one-fourth to one-third of embryos conceived result in a live birth. Data on embryo loss are difficult to obtain and estimates vary because its incidence is particularly high in the early stages of pregnancy when the loss is least easily recognized.

Assessment of individual reproductive function cannot be limited to evaluation of reproductive organs and reproductive cells because the many indices of reproductive health are closely tied to other physiological systems. Indices of impaired reproductive functioning include abnormal pubertal development, depressed libido, impotence, and irregular menstrual cycles. Physical examination should thus include assessment of circulatory, endocrine, and neurologic function. Patient histories should cover a broad range of factors that may influence reproductive health, including personal and family medical history, lifestyle factors, and work history.

The complexity of reproduction and development is mirrored by the complexity of the biological mechanisms that underlie toxic effects. These mechanisms involve absorption, distribution within the body, metabolism (toxification and/or detoxification), excretion, and repair.

A toxicant, whether a chemical, physical, or biological agent, acts by interrupting the normal function of a cell, tissue, organ, or organism. Reproductive toxicants may act directly in two ways. They may be structurally similar to an endogenous compound (hormone or nutrient) and thus mimic its action, or they may alter the structure of a hormone, causing it to vary in its activity. Toxicants may also act indirectly. Following metabolic conversion within the body, a secondary product acts on a tissue or organ of the reproductive system. Other toxicants act indirectly by altering the body's physiological control systems. Certain reproductive toxicants act in several ways simultaneously.

The toxicology of reproductive and sexual functioning is generally divided into two types: 1) reproductive toxicity, and 2) developmental toxicity. **A reproductive toxicant interferes with reproductive or sexual functioning of the adult from puberty through adulthood.** The many ways in which a reproductive toxicant can manifest itself include depressed libido, impotence, irregular menstrual cycles, and infertility. **A developmental toxicant produces an effect in the offspring from conception to puberty.** Developmental toxicity has four principal manifestations: 1) death of the conceptus, 2) structural abnormality, 3) altered growth, and 4) functional deficiency in the offspring. Some toxicants may have both reproductive and developmental effects.

Developmental toxicants can cause functional teratogenesis (alterations or delays in the postnatal abilities of the individual or delays in growth and development of organ systems), structural malformation, or altered growth. Developmental toxicants can act during either the embryonic or fetal periods, and can kill the embryo or fetus. These toxicants may be equally toxic to both parents and the embryo/fetus. The evolution of the concept of developmental toxicity and teratogenicity has implications for the language of TSCA, which refers to these substances as "teratogens," thereby implying the exclusion of substances that may cause other developmental effects. Modifying this language to refer to "developmental toxicants" would clarify the existing statute with regard to contemporary understanding of the word teratogen, since a teratogenic effect is one of several developmental effects.

EVIDENCE FOR WORKPLACE HAZARDS TO REPRODUCTIVE FUNCTION

By present-day standards, **there has been inadequate study of most suspected workplace hazards to reproductive function and procreative capacity in both men and women.** This situation exists for a variety of reasons:

1. Testing for workplace-induced reproductive impairment is a relatively recent phenomenon, stimulated in part by the thalidomide tragedy. In past years, studies were neither required by government nor considered necessary by industry. Thus relatively few of the thousands of chemicals used in the workplace have been evaluated for their potential effects on the reproductive systems of either animals or humans.
2. The effects of some hazards have been examined only in men and/or women, or in the developing offspring, but not in all three.
3. Many substances that have been tested for their toxic effects in animals have never been studied for their effects in humans, and more reproductive endpoints have been studied in animals than in humans.
4. Many study findings, particularly those of human effects, are inconclusive because of methodological problems.
5. Methods for extrapolating observed reproductive and developmental effects in laboratory animals to possible similar effects in humans are only now being developed.
6. Data on human exposure levels and particular endpoints that indicate reproductive impairment are difficult to obtain.

The scientific literature from human epidemiological and animal toxicology studies was reviewed for evidence of reproductive effects from exposure to a selected list of chemical, physical, and biological hazards, and to stress. The substances that were reviewed are listed in table 1-1. With the exception of certain metals (e.g., lead, mercury) certain organic solvents and pesticides (e.g., DBCP, EtO), ionizing radiation, and certain biological agents (e.g., rubella, mumps), **evidence linking particular agents with reproductive and/or developmental effects in humans is, for the most part, inconclusive.** Some substances have been studied more intensively than others, however. For example, anesthetic gases have been studied fairly extensively in humans, and major studies of the reproductive health effects of exposure to dioxin and prolonged use of video display terminals (VDTs) are currently in progress.

Table 1-1.—Agents and Substances Reviewed for Reproductive Health Effects by OTA

Metals:
- Lead
- Boron
- Manganese
- Mercury
- Cadmium
- Arsenic
- Antimony

Chemicals:

Agricultural chemicals:
- Carbaryl
- Dibromochloropropane (DBCP)
- DDT
- Kepone (Chlordecone)
- 2,4,5-T, Dioxin (TCDD), and Agent Orange
- 2,4-D

Polyhalogenated biphenyls:
- Polybrominated biphenyls (PBB)
- Polychlorinated biphenyls (PCB)

Organic solvents:
- Carbon disulfide
- Styrene
- Benzene
- Carbon tetrachloride
- Trichlorethylene

Anesthetic agents:
- Epichlorohydrin
- Ethylene dibromide (EDB)
- Ethylene oxide (EtO)
- Formaldehyde

Rubber manufacturing:
- 1,3-Butadiene
- Chloroprene
- Ethylene thiourea

Vinyl halides:
- Vinyl chloride

Hormones

Undefined industrial exposures:
- Agricultural work
- Laboratory work
- Oil, chemical, and atomic work
- Pulp and paper work
- Textile work

Physical factors:

Ionizing radiation:
- X-rays
- Gamma rays

Nonionizing radiation:
- Ultraviolet radiation
- Visible light
- Infrared radiation
- Radiofrequency/microwave
- Laser
- Ultrasound
- Video display terminals
- Magnetic field
- Hyperbaric/hypobaric environments
- Hot environments
- Cold environments
- Noise
- Vibration

Stress

Biological agents:
- Rubella
- Cytomegalovirus
- Hepatitis B
- Other infectious agents
- Recombinant DNA

SOURCE: Office of Technology Assessment.

Photo credit: Pemina Meisels

Reports of reproduction system effects among users of the many video display terminals (VDTs) now in use in the Nation's workplaces have raised questions about the safety of prolonged VDT exposure. Comprehensive studies of these effects are now in progress.

REPRODUCTIVE RISK ASSESSMENT

Risk assessment is the use of scientific evidence to estimate the likelihood of adverse effects on the health of individuals or populations from exposure to hazardous materials and conditions. Risk assessment is often confused with risk management, although the two are distinct. Risk assessment evaluates the probability of biologically significant events, while risk management determines the possible actions that can or should be taken in response to an assessment that a substance or condition poses a significant risk.

Several Government agencies are charged with the regulation of harmful substances. Because these agencies have different mandates based on the legislation underlying their authority and the types of substances and environments in their jurisdiction, the feasibility of centralizing risk assessment and management processes among them is uncertain. There is the potential, however, for establishing guidelines that can make these processes more explicit.

In risk assessment, no matter how clear-cut the evidence for the hazard, there are always scientific unknowns. It is not possible to predict the likelihood of a particular health effect from given exposure without some degree of uncertainty regarding the specific number of people who may be affected. Scientific decisions regarding the use of particular models or dose-response curves may carry with them judgments that generate different assessments of risk, and thus result in different risk management policies.

There are four steps in risk assessment: hazard identification, dose-response assessment, exposure assessment, and risk characterization.

- Hazard identification is the qualitative analysis of all available experimental animal and human data to determine whether and at what dose an agent is likely to cause reproductive or developmental effects. Hazard identification determines the potential of an agent to do harm, not the probability that harm will, in fact, occur.
- Dose-response assessment determines the relationship between the magnitude of human exposure and the probability of human health effects. In this step the results of animal studies, during which high doses are often given, must be extrapolated to effects on humans, who are usually exposed to smaller doses and vary with respect to exposure, susceptibility, and lifestyle.
- Exposure assessment identifies the population segments potentially exposed to the agent, including their composition and size, as well as the magnitude, frequency, and duration of potential exposure to the agent. This information is difficult to obtain because exposure can occur in different time patterns (acute v. chronic), or by different routes (inhalation v. skin contact), and exposure information on worker populations is often unavailable.
- Risk characterization, the final step, summarizes information about the agent and evaluates it in order to estimate the risk. An important component of this phase is estimating the level of uncertainty in the conclusions.

Most agents for which risk assessment is necessary are chemicals. Most of the 5 million known chemicals are probably not harmful at typical exposure levels. Many chemicals are manufactured in small quantities or are used in small amounts in research laboratories. For example, of the more than 48,000 chemicals* listed in the TSCA inventory (which lists substances in commerce but does not include pesticides, food additives, or cosmetics), only about 12,800 are manufactured in quantities of more than 1 million pounds per year, 13,900 are manufactured in quantities of less than 1 million pounds per year, and 21,700 are produced in unknown amounts. Workers are therefore unlikely to be exposed to more than a few of these chemicals in most workplaces. Because no publicly available toxicity information exists for more than 70 percent of the chemicals described in the TSCA inventory, it is currently impossible to evaluate their health effects.

Results from both animal toxicology and human epidemiology studies are used in the risk assessment process. Toxicology studies have several advantages. The experimental situation can be controlled, animals can be given specific doses in controlled environments, and results can **predict** the possibility that an agent is a reproductive health hazard in a particular animal. Their principal disadvantage lies in the necessity for extrapolation to human health effects. Adequate mathematical models for extrapolating dose-response curves from animal toxicology studies to human effects have not been developed. In addition, there is some biological basis for the assumption of threshold effects[3] in the developing embryo/fetus. Animal studies will continue to be necessary, however, as they provide essential information, and it is unethical to deliberately expose humans to potentially toxic substances.

Epidemiological studies may confirm an association between exposure to a hazard and reproductive impairment in humans. Unfortunately, once the effect is detected, the harm or damage has already been done. Epidemiology studies often suffer methodological problems because sample sizes of worker populations may be too small to significantly demonstrate effects on reproductive or developmental endpoints whose frequency is low in the overall population (e.g., congenital malformation). Many reproductive endpoints (e.g., spontaneous abortion, depressed libido) are difficult to measure. Some study designs have not controlled for the possibility of paternally mediated effects. Exposure is difficult to estimate and individuals may have lifestyle characteristics (alcohol, drug, or tobacco use) that confound study results. Moreover, workers, fearing loss of privacy, may be reluctant to cooperate in studies, and employers, fearing liability if results indicate evidence of harmful effects, may hesitate to conduct studies or to make data available to others for analysis.

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*1982 total; this figure now exceeds 63,000.

[3] The threshold concept assumes no harmful effects from exposure below a critical level at which no harmful effects are observed. By contrast, in cancer risk assessment, exposure to carcinogens is assumed always to present a risk, however low.

Federal agencies are concerned to varying degrees with reproductive risk assessment. The National Institute of Occupational Safety and Health (NIOSH), as the research and information support agency for the Occupational Safety and Health Administration (OSHA), is carrying out research on reproductive impairment, and is in the beginning phases of reproductive risk assessment. The Environmental Protection Agency (EPA) is carrying out research on reproductive impairment and is developing risk assessment guidelines on relevant topics. EPA's *Proposed Guidelines for Assessment of Developmental Toxicants* (in conjunction with three other proposed guidelines) has been published for comment in the Federal Register, and another, *Proposed Guidelines for Reproductive Risk*, will be completed in 1986. The EPA Developmental Toxicant guidelines assume the existence of thresholds and recommend the use of arbitrary safety factors for extrapolating safe exposure levels to humans until adequate mathematical models can be developed (see chapter 3). EPA is also completing Federal radiation protection guidelines that include recommendations for protection of workers from reproductive effects. The Nuclear Regulatory Commission (NRC) has also developed guidelines for protection of reproductive capacity.

REPRODUCTIVE RISK ASSESSMENT IN THE REGULATORY PROCESS

Occupational Safety and Health Administration

The OSH Act of 1970 gave the Federal Government responsibility for the occupational health of more than 75 million working Americans or some three-fourths of today's U.S. work force. OSHA, established by the Act, is the primary regulator of hazardous occupational exposures, including those that may cause reproductive effects.

OSHA has authority to regulate occupational health hazards in various ways. It may promulgate permanent or temporary standards, it may issue guidelines for employers when no standards exist, and it may enforce the general duty clause of the OSH Act.

- **Permanent Health Standards**. OSHA can promulgate permanent health standards for a single hazardous substance, for a group of specific substances, or even for a class of substances, but extensive and cumbersome rulemaking proceedings may take several years to complete. OSHA has promulgated permanent standards for three substances—DBCP, lead, and ethylene oxide—that include specific provisions for the protection of reproductive health.
- **Emergency Temporary Standards (ETSs).** OSHA may issue an ETS, effective immediately, if it determines that employees are exposed to a "grave danger" from exposure to a health hazard. No court has decided whether reproductive health problems are grave dangers, although a recent Federal court of appeals decision suggests that only "incurable, permanent, or fatal" health consequences could support the issuance of an ETS. Since OSHA has lost several challenges to its ETSs in the courts of appeals, OSHA is unlikely to issue ETSs for known or suspected reproductive health hazards.
- **Guidelines for Employers**. Even where no temporary or permanent health standards apply, OSHA may issue guidelines to employers to follow as an interim measure to protect workers while a standard is being set.
- **General Duty Clause.** OSHA is empowered

to ensure that employers are fulfilling their general duty under the OSH Act to furnish working conditions free from "recognized hazards" that are likely to cause death or serious physical harm. Because a hazard is considered recognized only if it is common knowledge in the employer's industry or if the employer had actual or constructive knowledge of the hazard, OSHA may not be able to prove that newly documented or suspected reproductive health hazards are recognized. In any case, OSHA rarely enforces the general duty clause at present. The general duty clause is therefore unlikely to substitute for an ETS as an interim measure until a permanent standard is enacted.

OSHA may not have the authority to regulate employment policies that exclude women from jobs that entail exposure to suspected reproductive hazards. The Occupational Safety and Health Review Commission ruled that Congress intended a "hazard" to be a process or material that causes injury or disease by operating directly on employees as they engage in work. This decision suggests, for example, that OSHA does not have authority to issue a citation to an employer on the grounds that its fetal protection policy itself constitutes a hazard even though the policy may result in women submitting to surgical sterilization in order to keep their jobs. In 1984, the Commission's decision was affirmed by the Federal court of appeals for the District of Columbia.

Even if OSHA could expedite the permanent health standard procedures or enact ETSs without fear of being reversed in court, health standards for reproductive health hazards might not result. Harmful substances are difficult to identify and interagency cooperation with NIOSH has varied with the political philosophy of the Administration in power. Under the Carter Administration, OSHA and NIOSH developed a close working relationship, including personnel exchanges and various joint programs, though this resulted in criticism of NIOSH for allegedly abandoning its research neutrality. The Reagan Administration, which believes in the clear separation of research (risk assessment) from regulation (risk management), has discontinued some cooperative programs.

OSHA also has a shortage of the professional and technical staff needed to develop health standards. This staff shortage may result in insufficient technical expertise to evaluate NIOSH's work and undertake appropriate regulatory actions.

Environmental Protection Agency

EPA has statutory authority under TSCA and FIFRA to regulate certain occupational exposures to reproductive health hazards, and under Executive Order No. 10831 to recommend Federal radiation protection guidance for workers. Like OSHA, EPA faces institutional and political uncertainties as well as scientific uncertainties that may constrain regulatory action.

EPA's administration of TSCA and FIFRA is constrained by data collection efforts that are not systematized enough to provide EPA with complete and consistent data for assessing reproductive effects of chemicals. Although TSCA requires companies to submit all available health effects data prior to manufacture of a toxic substance, testing rules do not address the full range of reproductive and developmental effects. New FIFRA regulations may begin to address a similar problem for pesticide manufacturers, who now, for the first time, are required to submit information on the potential reproductive effects of products regulated under FIFRA.

EPA has recently moved aggressively to take the regulatory lead from OSHA for substances that have potential health effects, including reproductive and developmental effects; e.g., benzene, ethylene oxide (EtO), formaldehyde, and glycol ethers. Public interest groups have persuaded EPA to yield to OSHA in regulating EtO, for example, because EPA does not have clear authority or resources to inspect or enforce EPA regulations in hospitals. EPA referrals to OSHA are likely to be made with increasing frequency.

EPA is, however, the primary governmental body regulating the hazardous exposure of farmworkers, whose working environment is very different from that of other workers. For example, unless drinking water is supplied, farmworkers may be forced to drink water from ditches or other open sources that may be contaminated with pesticide and herbicide residues. A proposal to include children under 12 years of age within farmworker protection standards because of their special vulnerability and because they "might be in the field at any time" was dropped in 1974 after strong protests from growers and their associations. Although some pesticide manufacturers label products suspected of being hazardous to pregnant women, EPA standards do not discuss whether pregnant farmworkers require special precautions, nor do public comments to the 1974 proposal indicate that the potential for reproductive effects among pesticide applicators (male or female) has received adequate attention.

No single agency regulates radiation exposure; Federal responsibility is dispersed among five executive departments, one independent commission and two agencies, and by diverse statutory provisions. Federal responsibility operates under the unifying force of Federal radiation protection guidance administered by EPA. EPA is revising the existing (1960) Federal radiation protection guidelines for workers. The guidelines will include specific provisions for protection of reproductive health and the health of the embryo/fetus. The currently recommended exposure limit of 3 rems per quarter (3 months) whole-body dose equivalent limit is expected to be reduced. Officials believe the new limits will be sufficient to protect against the risk of cancer and genetic effects. The draft also recommends that the policy of conforming to the lower limiting value for the developing embryo/fetus should be achieved without economic penalty or loss of job opportunity and security to the workers. The draft is to be transmitted to the President for approval in late 1985.

Nuclear Regulatory Commission

NRC regulations provide for some protection of reproductive health. The regulations provide for maximum exposure levels, including limitations on exposure to gonads and lifetime cumulative dose, and protection of the biological systems of minors. **There are no provisions that deal with protection of the embryo/fetus or with pregnancy per se, although some expert groups have recommended reduction of exposure limits for fertile and pregnant workers. Other expert groups have argued for a gender-neutral policy that protects male and female workers from mutagenic risks.**

The nature of the regulations promotes the use of temporary employees. These workers generally receive higher doses over short intervals than do regular workers. Temporary workers constituted 35 percent of the work force in the nuclear power industry in 1977, but received an estimated 47.5 percent of the total work force radiation dose.

The factual basis for NRC health regulations has not been adequately tested in the courts. Federal courts have repeatedly deferred to NRC expertise and discretion.

SEX DISCRIMINATION

Some companies and health care facilities have implemented, or are considering, policies that exclude women of childbearing age or capacity from jobs involving exposure to suspected reproductive or developmental hazards. Although it is impossible to determine how many companies have either written or unwritten exclusionary policies, **at least 15 of the Fortune 500 as well as numerous hospitals are reported to exclude fertile and/or pregnant women from some jobs.**

Company exclusionary policies vary greatly. Some are based on epidemiological and toxicological research findings with respect to particular substances; others are relatively speculative about suspected reproductive hazards. Some policies are carefully written and documented; others are unwritten, making them more flexible but also more ambiguous. In large manufacturing companies, policies are generally announced to employees and their unions, if applicable, prior to implementation; smaller organizations appear to formulate and apply policies as a perceived problem arises. Some policies recognize that a developmental hazard may be mediated through either male or female workers, while others apply only to women.

In some cases, these policies have faced court challenges on grounds of sex discrimination in violation of Federal law. **Title VII of the Civil Rights Act of 1964 prohibits employment discrimination on the basis of sex, while the Pregnancy Discrimination Act of 1978, an amendment to Title VII, specifically forbids discrimination on the basis of pregnancy, childbirth, or related medical conditions.** The amendment requires that women affected by these conditions be treated the same for all employment purposes as others not so affected but similar in their ability or inability to work.

While many of these cases are apparently settled out of court, some have been adjudicated and three have been reviewed by the Federal courts of appeals in the Fourth, Fifth, and Eleventh Circuits. **All three courts have held that the exclusion of fertile or pregnant women due to the existence of alleged hazards to the embryo/fetus is permissible if scientifically justified and if less discriminatory alternatives do not exist. In all other circumstances, such exclusionary policies constitute illegal sex discrimination.** Although the three courts used different approaches, the following general principles can be extracted from these cases:

> A fetal protection policy (FPP) that applies only to women is presumptively discriminatory. That is, the mere existence of an FPP will create Title VII liability for the employer in the absence of strongly supportive scientific evidence.
>
> To overcome the presumption of discrimination, the employer must be able to present persuasive evidence that the body of scientific evidence supports legal findings that: 1) exposure at the level encountered in the workplace involves a significant risk of harm to the unborn children of women employees, 2) exposure at the level encountered in the workplace does not involve a similar risk of harm to the offspring of male employees, and 3) the FPP is effective in significantly reducing the risk. An employer's subjective but scientifically unsupportable belief in the necessity of the policy is insufficient to defend it.
>
> If the employer proves both points (embryo/fetal risk through maternal exposure and lack of embryo/fetal risk through paternal exposure), the plaintiff may nevertheless prevail by proving that an acceptable alternative policy would promote embryo/fetal health at least as well with a less adverse impact on one sex or by showing that the FPP is a pretext for discrimination.

WORKERS' COMPENSATION

The primary goal of workers' compensation is to provide relatively rapid and fair compensation for workplace-induced accidents or illnesses. Workers' compensation laws (and, to some extent, tort law) are also intended to deter hazardous conduct by employers through the use of economic disincentives, based on higher insurance costs and/or more frequent payments to injured workers. OSHA and other agencies with the authority to mandate workplace conditions were created in part as a response to the failure of workers' compensation laws to have a significant deterrent effect. **Both the workers' compensation and tort liability systems fail to consistently provide compensation to the victims of occupationally induced reproductive impairment, though they sometimes result in some compensation for some workers.** Few workers seeking workers' compensation on the basis of reproductive impairment would be able to meet the following three criteria for eligibility, which state that the injury or disease must:

1. Be a *"personal"* injury or disease. This would preclude compensation for injuries or diseases suffered by others, such as the worker's spouse, fetus, child, or descendant.
2. Result in *job disability*. This requirement would prevent the award of disability benefits for most claims of reproductive injury or disease, since such harms do not usually disable the worker or prevent him or her from resuming work at the same job.
3. Be *caused* by a workplace accident or ex-

posure: Proving causation is difficult. Workers' compensation boards generally prefer medical evidence that a particular individual contracted a particular disease in a particular way to scientific evidence that shows how many, or even most, people contract the disease. The causation problem is endemic to occupational disease claims in general.

A few State systems utilize a "whole body" concept of disability that covers personal injuries that do not prevent a worker from returning to work. These States may allow reproductively impaired workers to collect a scheduled benefit, although only one State has considered the issue. The effects of the eligibility criteria on workers are summarized in table 1-2.

Because the "exclusivity of remedy" doctrine embedded in most workers' compensation statutes provides that an employee covered by such statutes cannot sue his or her employer at common law for any injury or disease subject to the worker's compensation statute, workers are often barred from seeking common law remedies. This bar to worker suits has generally been maintained by the courts *without regard to whether the worker's claim actually resulted in the payment of benefits.*

If workplace exposure is determined to have adverse reproductive effects, workers presently have no remedies or, at most, inadequate remedies in the workers' compensation systems of most States. These victims of hazardous occupational exposures will, by default, bear the burden of their occupational exposures to reproductive health hazards.

Table 1-2.—Summary of Harms, Victims, Benefits Criteria, and Causation Problems in Workers' Compensation Systems

	Victim		
Circumstances of harm	Worker	Spouse	Embryo/fetus and offspring
1. Accidental injury to worker reproductive system or embryo/fetus resulting in injury or disease to a part of body covered by schedule or in loss of work	Personal injury: eligible for compensation for medical benefits in all States and loss of function and disfigurement in a few States. No disability unless earnings loss. No special causation problems	Not personal injury, therefore no compensation	Not personal injury, therefore no compensation
2. Acute or chronic exposure of worker, spouse, or embryo/fetus	If personal injury, will be eligible for compensation for medical benefits in all States and loss of function benefits in a few States. No disability benefits unless earnings loss. Special causation problems	Not personal injury, therefore no compensation	Not personal injury, therefore no compensation
3. "Side effect" cases where reproductive function impaired due to other diseases	Probably not applicable since other injury or disease will be primary personal injury for disability compensation, not the reproductive injury	NA	NA

NA—Not applicable.

SOURCE: Office of Technology Assessment.

TORT LIABILITY

The body of law governing personal injuries is known as tort law. Perhaps more than any other area of the common law, tort law is a battleground of evolving social theory.

Workers alleging reproductive injury may bring lawsuits against two primary types of defendants. First, they may try to sue their *employers* for alleged negligence, intentional tort, strict liability, or product liability. Second, they may bring suit for negligence, strict liability, or product liability against the *manufacturers* of products used in the workplace that may have caused or contributed to the injury or disease.

Although the exclusivity rule operates to bar worker tort suits against their employers, two principal arguments have proven effective in convincing judges to allow suits against employers in some jurisdictions: the dual capacity exception and the intentional tort exception.

Dual capacity exists when the employer is also a manufacturer of the product that caused the worker's injury or provides medical services for the injury in a negligent fashion. Although some States allow an injured employee to sue a dual capacity employer, this exception has been opposed by industry and has been rejected in 23 States. Under the intentional tort exception, evidence that an employer's conduct manifested a deliberate attempt to injure a worker can also be used by the worker to overcome the exclusivity rule and bring a tort action against the employer. However, the fact that an employer's conduct is egregious is usually, in itself, insufficient to prove deliberate intent to cause injury. **Therefore, for the most part, reproductively damaged workers have very limited access to redress against their employers through the courts.**

Suits against employers or product manufacturers may be brought not only by the injured worker but also by others who may have been injured. One type of potentially injured party is especially relevant to reproductive health hazards: the embryo or fetus that has not been born, perhaps not even conceived, at the time the hazardous exposure occurs. The controversy over the rights of the affected child to recover for prenatal and pre-conception injuries has increased dramatically over the last 40 years. Where once there was complete denial of any rights, the courts now grant recovery in almost every situation resulting in injury to an embryo/fetus who is eventually born alive. Although these cases generally involve negligent medical treatment, the basis for liability to an embryo/fetus does not appear to be limited to medical malpractice. The extent of these legal rights varies greatly among jurisdictions, however, as courts struggle with the unique problems posed by the unresolved status of the embryo/fetus. **Although all States now recognize the right to bring an action for prenatal injuries, many jurisdictions will deny recovery unless the fetus has reached the stage of viability when it is injured. In these jurisdictions, lawsuits for many developmental effects, such as birth defects resulting from chromosomal aberrations or embryo toxicity, would not be permitted because the injury occurred prior to viability.**

ETHICAL CONSIDERATIONS

The management of exposure to reproductive and developmental toxicants in the workplace presents ethical dilemmas because a course of action that may be justified by ethical principles can carry with it both desirable and undesirable consequences.

Reproductive health hazards in the workplace raise ethical issues in three areas. First, the management of suspected hazards often focuses on women workers, who traditionally have been discriminated against under the guise of protecting their reproductive health or the health of their offspring. Second, there is the equivocal status of an embryo/fetus who cannot consent to the risks that may be involved. Third, reproduction is one of the most sensitive and intimate aspects of life, which raises issues of worker privacy.

The ethical principles most relevant to the issues of exposure to reproductive health hazards in the workplace are: 1) respect for persons, 2) beneficence, and 3) justice.

Respect for Persons

The principle of respect for persons requires that individuals be treated as the focus of concern in their own right and not merely as the means to the achievement of other goals. This principle has important applications both for workers exposed to reproductive hazards and for their offspring and potential offspring. Respect for persons requires informed and voluntary choices by individuals about matters that affect their well-being and life prospects. Informed choice by workers implies a duty on the part of employers and unions (and possibly the government) to disclose existing information about reproductive health hazards in the workplace. Voluntary choice based on accurate information allows workers to maintain their autonomy.

The principle of respect for persons offers little real guidance on the specific duties of employers towards workers' offspring and potential offspring. The difficulty lies in the fact that ethically and legally, fetuses, infants, and even young children have an equivocal status as "autonomous" beings. In general, the interests of fetuses, infants, and children fall more naturally under the principle of beneficence, since all persons and potential persons are entitled to benefits and protection from harm.

Beneficence

The principle of beneficence requires avoiding harms to others and maximizing the balance of benefits over harms. Beneficence is a consideration in at least three relationships in the workplace: employers' duty to workers, workers' duty to offspring, and employers' duty to offspring.

Employers' Duty to Workers

The specific and general legal duties specified under the OSH Act imply an ethical duty to avoid exposing workers to unreasonable risk of harm. The OSH Act may be a statutory codification of an evolving social conviction that the duty exists at the moral level. The Civil Rights Act implies a corresponding duty not to discriminate in the employment opportunities of individuals.

Workers' Duty to Their Offspring and Potential Offspring

Parents may have certain duties to the expected child even while it is an embryo/fetus. Such duties might equal but could not exceed the duties owed to newborn infants. This points up a limitation to the duties owed embryo/fetuses: beneficence requires one to do what is best, on balance. It is not a duty to avoid any and all possible harms to the embryo/fetus when that same action might gain some benefits to the embryo/fetus and avoid other harms. From the standpoint of the management of exposure to reproductive health hazards, a parent who chooses to continue working in a mildly hazardous workplace is not necessarily violating any duty of beneficence to his or her embryo/fetus. For example, the benefits of working in a mildly hazardous situation might include improved prenatal health care, and better housing and food.

Employers' Duty to Workers' Offspring and Potential Offspring

The scope of employers' duty to their workers' embryo/fetuses is difficult to determine because of the lack of a clear relationship between employer and embryo/fetus, and ambiguities in the moral status of an embryo/fetus. While the worker-parent's exposure is to some degree voluntary, the fact that the embryo/fetus has not "consented" to be exposed to hazards should not automatically lead to the implementation of a higher standard of protection for the embryo/fetus than for the worker-parent, unless the embryo/fetus is more susceptible.

This underscores the interaction of the principles of respect for persons and beneficence: **the duty to protect certain persons or embryo/fetuses from harm may be in conflict with the duty to permit other persons maximum latitude for free and informed choice.**

Justice

Justice is the fair and equal treatment of others. This principle is relevant to the management of reproductive health hazards in at least two ways: 1) the differential impact on male and female workers, and 2) the allocation of burdens.

Differential Impact on Male and Female Workers

The principle of justice requires that like cases be treated alike. Thus policies that have a heavier negative impact on workers of one sex may not be just unless the cases are not alike. Fetal protection policies have typically been directed to women, who are much more likely than men to be removed from or denied jobs on the grounds that reproductive or developmental hazards exist. Unless such policies are based on relevant and important differences, they can be regarded as unjust.

Allocation of Burdens

There are two burdens to be allocated: financial burdens and health burdens. Generally, serious impairment to a person's health is perceived as a greater harm to that person's interest than are financial burdens, especially when financial burdens are spread over a large number of individuals, with little impact on each.

ISSUES AND OPTIONS

In many ways, reproductive health hazards are like other occupational health hazards. There is scientific uncertainty about the health effects of most occupational exposures. What should society's decisionmakers—employers, workers, regulatory agencies, courts, and legislators—do in the face of such uncertainty? What should be assumed about risk when it is unclear whether a substance is hazardous or not? What are the costs to the affected groups and to society in general? How can risks, expenses, and other burdens be apportioned fairly?

When these questions are asked in the context of the management of exposure to reproductive health hazards, however, it is important to consider this salient difference: men and women are physiologically distinct, especially with respect to reproduction. Are their biological differences of such nature and magnitude as to require differential treatment? Again, scientific uncertainty about the effects of chemical, physical, and biological exposures obscures the answer. Reproductive health hazards are also different because they can affect the offspring as well as the adult. This reality presents moral and legal questions about who is entitled to make certain decisions that may affect the health and well-being of future generations.

This discussion of the policy issues and options begins with an issue that is unique to reproductive health hazards in the workplace: the use of sex-based employment policies that exclude female workers from workplaces containing suspected reproductive and/or developmental hazards. Issues that are not confined to reproductive health hazards, such as general occupational and environmental disease problems concerned with prevention, regulation, and compensation in the face of scientific uncertainty, are then summarized.

Sex Discrimination

Because of scientific uncertainty, it is difficult for an employer to meet the three criteria for justifying fetal protection policies (FPPs) that exclude only female (fertile or pregnant) workers from jobs involving exposure to suspected developmental health hazards. The mere existence of an FPP that applies only to women will, in the absence of strongly supportive scientific evidence, create liability for illegal sex discrimination under Title VII of the Civil Rights Act.

For those chemical, physical, and biological agents that have been researched for human reproductive effects, scientific evidence generally fails to confirm or disconfirm a need for differential exposure standards for men and women based on either reproductive effects on the adult or parentally mediated effects on future offspring. This is because most suspected hazards have not been thoroughly researched for their reproductive effects in both males and females and for developmental effects in the offspring.

In the face of scientific uncertainty about many of the chemical, physical, and biological agents to which American workers are exposed, and with the great publicity given to substantial personal injury verdicts in product liability cases, employers feel obliged to take action to protect their employees and their future offspring, and to defend their own economic interests.

The tort system provides incentives to employers to abate hazardous conduct. However, the employer's economic interests are much greater with respect to developmental hazards (those that affect the embryo or fetus due to parental exposure before conception or maternal exposure after conception) than they are for other reproductive hazards. For reproductive impairment, most State workers' compensation schemes both fail to provide compensation for the victims of occupationally induced reproductive and sexual impairment and prohibit employee personal injury lawsuits against employers. For developmental injuries, however, the offspring of exposed workers would not be covered by workers' compensation and therefore would have a right to sue the parent's employer. In addition, the harm that could be done to an embryo or fetus could be permanent and devastating, and could result in heavy liability, while effects on adult sexual or reproductive function, while potentially personally devastating or physically damaging, are unlikely to be physically or occupationally disabling and may be reversible.

Congress could consider whether the employer's greater economic incentive to prevent exposure to developmental hazards (as opposed to hazards to adult reproductive function) is justified by ethical or public health considerations: should the health of potential children be protected to a greater degree than the health and well-being of their parents?

Exposure to developmental hazards can occur either prior to conception or during pregnancy. Prior to conception, exposure may result in damage to a male worker's sperm cells or a female worker's egg cells. During pregnancy, exposure to a developmental hazard can be maternally mediated. There is also the possibility that an exposed man may transmit exposure to his pregnant wife who in turn exposes the embryo/fetus.

Officials in many companies believe that effects on future offspring are most likely to be caused by direct exposure of the pregnant woman, rather than by exposure of either parent prior to conception or by exposure of the sexual partner of a pregnant woman. This is, in part, true because of the relative abundance of animal studies of developmental effects on the embryo/fetus due to exposure of pregnant females. There is a corresponding dearth of scientific information concerning possible male-mediated effects. Since companies anticipate being held financially and morally liable should fetal injury occur, many feel forced to employ only males in certain workplaces in order to avoid potential liability to a damaged infant. **Since there are no records of any lawsuits brought by the children of exposed women workers, critics of industry policies suggest that fear of liability is speculative.** To the extent that such liability might exist, some critics note that it could extend equally to the offspring of male workers.

Employers have a range of options, each with limitations. Further reducing exposure or eliminating the suspected hazard is the most effective and least discriminatory option, but may be the option with the highest cost and may not be economically or technologically feasible for particular employers or substances. In other cases reducing exposure to safe levels may be impossible because too little is known about the hazard to establish a no-observed-effects-level (NOEL). Nevertheless, reducing exposure or eliminating the hazard may be cost-effective overall, when society's costs and benefits are added to those of the company.

Monitoring female workers for pregnancy, even if scientifically and legally defensible, would involve considerable intrusion on personal privacy and be difficult to implement. Monitoring is also likely to be only moderately effective because pregnancies are often not known or disclosed before exposure occurs and because no prevention of possible male-mediated effects would result. Voluntary medical removal policies for employees who are planning to parent children are less burdensome on workers and minimize differential treatment of men and women if applied to both sexes. However, if a pregnancy is unplanned, voluntary removal may not have occurred early enough to prevent injury.[4]

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[4] Among women age 15 to 44 in the labor force in 1982, 33.6 percent of births in the previous 5 years were unplanned (7.6 percent were unwanted and 26.0 were mistimed). These data do not indicate whether these women were working at the time they became pregnant. (W. Pratt, personal communication, 1985, tabulations from the National Survey of Family Growth (NCHS), 1982).

The option of using sex-based distinctions in hiring and assigning workers, and then attempting to defend in court, is risky: the science and the law are in flux, and such exclusionary policies may be rejected due to corporate concerns about fairness or reputation. Nevertheless, sex-based distinctions may be less costly than other options for some employers, notwithstanding possible court challenges. Finally, various options involving personnel and medical counseling can be used to promote voluntary removal policies or coerce involuntary removal of female workers. An employer may find that one or more of these options protects his or her interests, though not necessarily those of his or her employees.

These options may be viewed as falling on a continuum from being more protective of embryo/fetal health and less protective of employment rights to less protective of embryo/fetal health and more protective of employment rights. In many cases, this is an oversimplification, since options that protect against paternally mediated effects may increase protection of the embryo/fetus while spreading the burdens more evenly between men and women. Nevertheless, most options can be classified as either overprotective or underprotective, and the issue is whether the price of either is too high.

OPTION 1:
Congress could maintain the status quo.

Congressional inaction would effectively continue the existing system of employer flexibility in tailoring fetal protection programs to existing scientific information concerning risk. As discussed above, the courts have set guidelines under which certain sex-based employment distinctions are permissible under Title VII when risks to the embryo/fetus are involved. If the status quo is maintained, any evolution of the law in this area would take place in the courts.

Maintaining the status quo also maintains the financial incentives: an employer might anticipate that the expense of losing a sex discrimination lawsuit would be smaller than the verdict in a single lawsuit brought by the offspring of a worker for personal injuries sustained in utero. This suggests that, notwithstanding Title VII's prohibition, sex-based distinctions may be the favored alternative in some cases, even where they are not scientifically supportable.

OPTION 2:
Congress could amend Title VII so as to prohibit FPPs that apply only to women unless scientific evidence exists showing that there are no paternally mediated effects.

Research on reproductive health effects of various substances has focused on female-mediated developmental effects in human and animal populations and generally overlooked the possibility of male-mediated developmental effects or other reproductive effects. This bias may be reflected in employment policies that exclude women from the workplace based on scientific data but allow men to remain exposed because of a lack of data concerning male reproductive health effects. Current scientific evidence is in most cases inadequate to determine the extent to which a substance that is hazardous to one sex may or may not be hazardous to the other.

Congress could therefore provide greater protection to the future children of exposed men and perhaps, over time, even reverse this research bias by amending Title VII to create a legal presumption concerning the scientific data in Title VII sex discrimination suits. The law could provide that any substance proven or suspected of being a hazard to one sex (or its future offspring) for the purpose of an exclusionary policy will be legally presumed to be a hazard to the other sex (and its future offspring) at similar exposure levels until substantial scientific evidence demonstrates the contrary to be true. This approach would help ensure that women's employment rights are not easily overridden. It would provide greater protection to men and their future offspring in cases where a substance is known to be harmful to women and their future offspring but where the evidence concerning men is not yet available. It would also encourage employers to undertake more scientific research on both male and female reproductive and developmental risk so as to be able to scientifically support a single-sex exclusionary policy. Finally, it would enable Congress to articulate how much scientific justification is necessary to support an employment policy that discriminates between men and women.

This option could make exposing both men and women economically preferable to excluding both, however, especially for small companies that cannot afford the research that would be required to overcome the legal presumption of similar effects on both sexes. An unpredictable number of embryo/fetuses could be exposed to hazards that are real but insufficiently documented to be the subject of a legal FPP that applies only to one sex.

This option might also discourage employers from engaging in any research at all if the result is likely to be the exclusion of men as well as women, or only men. Employers might decide to take the chance that a substance is harmful and could injure a worker's offspring rather than pay for research that might result in the expense of redesigning a workplace that would otherwise pose significant risks to both sexes.

While the current system may also result in an unpredictable number of paternally mediated developmental effects, this option could result in an unpredictable number of paternally *and* maternally mediated developmental effects. A similar proposal by the Equal Employment Opportunity Commission (EEOC) and Office of Federal Contract Compliance Programs (OFCCP) was withdrawn in 1981 due to these concerns.

In addition, Congress could make sex-based distinctions a less attractive employer option by providing an additional financial disincentive, such as recovery of punitive or treble damages by losing defendants in sex discrimination lawsuits. Such disincentives would also make it easier for employees who have been discriminated against to find lawyers willing to handle their cases.

OPTION 3:
Congress could require that employers with unproven but suspected developmental hazards in their facilities fully inform workers and allow individual employees to decide whether or not to continue in jobs involving such exposures. Employees would then be responsible for the consequences of exposures to which they consented.

An employer disclosure requirement could be coupled with employer immunity from personal injury suits should injury to an employee or his or her offspring result from the employee's informed consent to the exposure. Because it appears that a worker cannot legally waive his or her offspring's legal right to avoid injuries caused by developmental hazards, employers are generally unwilling to accept a worker's attempted waiver of the future offspring's rights. Under this option, if an employee were to decide to continue in a job involving exposure to a suspected but unproven developmental hazard, the employee would be legally, financially, and morally responsible for injury to his or her offspring. A possible suboption would grant employees the right to temporarily and voluntarily work at another job.

The major beneficiaries of such a policy would be employers, workers who do not parent children during the period of exposure or bioaccumulation (e.g., workers who practice sexual abstinence or who have undergone sterilization), and workers who parent healthy children because speculation about a suspected hazard was incorrect. Employers would benefit because they could avoid the economic burdens associated with the other options, as well as the potential expense of compensating damaged children. Workers who cannot or choose not to parent children would be free to expose themselves to suspected developmental toxins rather than be excluded from the workplace on the assumption that they might parent children.

There are several problems inherent in this option. The public health problem is that some employees may assume the risk, either because of scientific uncertainty, because they mistakenly believe the exposure will not hurt them, or because they are not planning parenthood, and produce injured children as a result. While workers intending to reproduce might not intentionally expose themselves to suspected developmental hazards, accidental pregnancies could have serious consequences for the health of the offspring. In these cases, this option may force a worker and his or her partner to choose between an abortion and an injured child. The public health problem could in fact extend beyond the injured children themselves and, in the case of genetic mutations, affect the health of future generations.

It is also questionable whether full disclosure or true informed consent can really be made in such circumstances. Technical information that is disclosed but not fully understood may lead to misinterpretation of the extent of risk. Furthermore, the prospect of unemployment or a wage decrease may leave the worker with little choice but to continue employment in a potentially hazardous workplace. These situations cast doubt on the concept of freely given consent. In addition, many people believe that shifting the burden for workplace risks to the employee is never ethical.

There is also an ethical issue as to whether a worker should be permitted to waive the rights of future offspring to be uninjured (or, if prevention fails, to be compensated for a job-induced injury), so that the worker can pursue his or her employment in a particular job and facility. Moreover, while it may seem fair to eliminate the employer's liability to the child of a worker who consents to exposure, the worker may not be financially able to assume the consequences of his or her decision, in which case this burden falls on society.

The financial benefit to employers may be minimal. Because of the scientific uncertainty involved, an employer's disclosure and an employee's consent will often be less than fully informed. In these cases, the worker and his or her injured offspring may attempt to bring a personal injury suit against the employer and have the worker's consent declared legally ineffective. Thus, employers may be subjected to the same legal battles and expenses that accompany the prophylactic use of exclusionary fetal protection policies.

SUBOPTION:

Congress could allow workers to temporarily and voluntarily remove themselves from jobs involving exposure to suspected reproductive health hazards.

OSHA provisions allow medical removal for employees exposed to some health hazards, such as lead.

In cases where the employee's uptake of the hazard can be easily measured, an employee could consent to be regularly monitored for his or her uptake of workplace substances until the concentration of suspected or known hazards was sufficiently elevated to warrant the employee's removal from that job. This monitoring could be limited to those who are trying to parent children or could be extended to all workers with reproductive capability. In cases where the employee's uptake cannot be measured easily, an employee who is trying to parent a child could voluntarily remove himself or herself from a job involving a potentially hazardous exposure. In many cases, however, measurement of exposure levels or safety levels cannot be accurately determined.

Upon removal from the job and its risks, the employee could be temporarily placed in a job without exposure to suspected reproductive or developmental hazards, either retaining the former wage rate or assuming the generally lower wage rate of the less hazardous job.

In cases where the employer could not economically justify placing the employee in another, nonhazardous position (e.g., where all such positions are filled, or where they require extensive training or education), the employee could be permitted to take a paid or unpaid leave of absence without losing seniority, health benefits, and/or eligibility for unemployment insurance or workers' compensation coverage during or after the period of absence. This option may not be realistic for many small businesses.

In a Pennsylvania case, involuntary removal from a job to protect worker health, including reproductive health, from further absorption of lead, and subsequent placement of the workers by the employer in different, lower paying jobs resulted in a successful claim for partial disability benefits. In reversing the Pennsylvania board's order denying benefits, the Pennsylvania Supreme Court stated:

> It would be barbaric to require an employee to continue in a position where he is exposed to a toxic substance until he is so ill that he is physically incapable of performing his job. We have held that . . . the word disability is to be regarded as synonymous with loss of earning power.[5]

Conceivably, this view could be extended to situations from which the employee voluntarily withdraws to avoid a reproductive health hazard with compensation to be provided for any resulting decrease in earnings. This policy would be comparable to cases of voluntary removal from health risks where the worker was not barred from securing unemployment benefits.

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[5] Lash v. Workmen's Comp. App. Bd., 420 A.2d 1325 (Pa. 1980).

After the voluntarily rotated or absent employee has parented a child (or determined that he or she is not able to parent a child), within a maximum timeframe designed to protect the employer, the employee could be allowed to resume his or her former responsibilities without penalty.

OPTION 4:
Congress could amend Title VII to explicitly permit FPPs that treat male and female workers differently when scientific information supporting differential treatment is inconclusive.

This protective public health approach offers greater protection to the embryo/fetus than some of the other options. It assumes that the embryo/fetus is more susceptible to workplace health hazards than are adults. This option also assumes that most injuries are maternally mediated during pregnancy and overlooks the possibility of damage due to pre-conception exposure of either father or mother.

Unfortunately, this option could permit unnecessary discrimination against female workers. In any given year, only 1 of 15 women aged 16 to 44 gives birth to a live child,[6] though all 15 might be subject to exclusionary policies that deny them their jobs or encourage them to submit to surgical sterilization due to speculation about risk of developmental effects. Furthermore, it is reasonable to assume that some of the substances for which scientific evidence is inconclusive are not in fact harmful to the embryo/fetus at the level of exposure encountered in the workplace. The level of protection to the embryo/fetus provided by this option would not reduce the risk of paternally mediated effects and could come at a substantial cost to female employment opportunities.

Regulation

Regulation in the Face of Uncertainty

Regulatory agencies such as OSHA, EPA, and NRC often face scientific uncertainty about whether a particular exposure constitutes a hazard to reproductive health. This problem exists for all areas of health regulation. Activities in the face of scientific uncertainty vary among Government agencies. Due to differing statutory mandates, OSHA, EPA, and NRC have developed their own procedures for corporate notification of new evidence concerning adverse health effects and agency response to toxicity information.

Should an agency regulate exposures when scant evidence suggests a possible health hazard, on the premise that worker health should be protected from all suspected hazards despite the substantial cost of such protection? Or should an agency only regulate when "all the evidence is in"—i.e., when there is a preponderance of evidence that a substance is harmful? OSHA, the agency charged with protecting occupational health, currently declines to regulate unless there is a preponderance of scientific evidence demonstrating the existence of a significant health risk.

OPTION 1:
Congress could maintain the status quo.

Agencies could continue to regulate exposures only after substantial evidence supports reducing exposure limits because of the finding of significant risk, even though this may result in more harmful exposures than might otherwise be the case. Regulating only when supported by substantial evidence would nevertheless serve to protect society from well-documented hazards while avoiding the costs associated with regulating suspected substances that later prove to be nonhazardous.

OPTION 2:
Congress could instruct the regulatory agencies to be more willing to assume that an exposure is dangerous when only a small number of studies suggest this.

Such an option would probably require a legislative amendment to the OSH Act specifying that an OSHA determination as to risk is conclusive if some evidence of risk exists. Such determinations might concern whether an observed health effect is occupationally induced or not, or whether evidence demonstrates an effect on animals but is only suggestive in humans. This would better enable OSHA to regulate when the scientific evidence is not substantial. Presently, a court can strike down OSHA regulations if the court believes there is not "substantial evidence" to support the standard.

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[6] On average, less than 1 of the other 14 women will have a pregnancy that fails to result in a live birth. Fetal loss may be attributable to exposure to occupational and/or other health hazards. In addition, some of these women will be exposed to hazards that are not manifested until a pregnancy 1 or more years later.

This option may result in great costs for "protection" from substances that are later shown not to be harmful at levels encountered in the workplace, but it could also protect some workers from exposure to a substance that is later, more conclusively, proven to be harmful.

Private Right of Action

OSHA is enforced solely by the Federal Government, except where States have federally approved State plans. Individual workers have no explicit right to go to court to force OSHA to issue citations to particular employers who are violating the Act. Thus, even if an employee has evidence that his or her employer is exposing him or her to a known reproductive (or other) health hazard, the employee probably cannot force OSHA to cite the employer either for violating an OSHA health standard or for violating the general duty clause.

OPTION 1:
Congress could maintain the status quo.

Congress may use its oversight and appropriations authority to maintain a level of OSH Act enforcement that is satisfactory to the Congress.

OPTION 2:
Congress could amend the OSH Act to grant employees the right to force OSHA to take action against employers who may be violating either an OSHA standard or the general duty clause.

This would enable workers to force OSHA to inspect a facility if there are reasonable grounds for concern about workplace health and safety hazards and to issue a citation if a workplace is found to be unhealthful or unsafe. Unless OSHA is provided additional funding and manpower for responding to worker petitions, however, the agency's resources may be diverted from other matters identified by administrative and scientific personnel as having higher priority.

Additional Relationships Between OSHA and NIOSH

Congressional action might help to protect workers from potential occupational health hazards by creating additional relationships between OSHA and NIOSH that enable or encourage OSHA to act on NIOSH-generated data about reproductive health hazards.

OPTION 1:
Congress could maintain the current relationship between OSHA and NIOSH.

Though the two agencies have common goals—the protection of occupational health in America's workplaces—their separation in the bureaucracy may sometimes result in lack of communication and thus a lack of compatible research and regulatory priorities.

OPTION 2:
Congress could join OSHA and NIOSH organizationally.

Although creating a single agency from the two might enhance communication and cooperation in risk assessment and risk management activities, either agency's removal from its current parent agency might compromise the quality of those activities. NIOSH's relationship with the Centers for Disease Control enables it to play an important role in the Federal Government's public health effort, while OSHA's relationship with the Department of Labor may make the agency more politically responsive than NIOSH. OSHA's Chief reports to a member of the President's Cabinet while NIOSH's does not; this may or may not affect agency interactions. The fact that different subcommittees of Congress oversee the activities of the two agencies does not help to increase coordination of priorities.

OPTION 3:
Congress could give NIOSH the power to force OSHA to respond to NIOSH recommendations concerning reproductive and other occupational health hazards.

When NIOSH evaluates suspected health hazards and makes recommendations to OSHA concerning regulation, OSHA is not presently required to respond. Congress could force OSHA to respond to NIOSH research and recommendations by requiring OSHA to act within a fixed time limit after receiving NIOSH research results and recommendations and either proceed as recommended or publish an explanation in the Federal Register of why such action would be inappropriate. This would place a burden on OSHA to articulate its reasons for failing to adopt health standards recommended by NIOSH.

The disadvantage of this option is that requiring OSHA to respond to NIOSH recommendations may dilute its personnel resources and prevent OSHA from attending to matters it considers more pressing. For example, a NIOSH study that finds that a particular substance may cause transitory infertility and that results in a NIOSH recommendation for regulatory action could require a formal OSHA response based on scientific, economic, and other data. Given OSHA's small technical staff, the legally mandated response to NIOSH and the public could prevent OSHA from investigating other suspected hazards that, while not yet the subject of completed NIOSH research, appear to be more hazardous. In addition, forcing OSHA to respond to NIOSH recommendations might dilute OSHA's ability to enforce existing standards.

Emergency Temporary Standards

Even when the evidence appears to strongly support a health standard, OSHA may not promulgate an emergency temporary standard (ETS) unless a "grave danger" exists. The Fifth Circuit Court of Appeals interprets this language to mean a danger of "incurable, permanent, or fatal consequences to workers, as opposed to easily curable and fleeting effects on their health." Given this definition, some reproductive health hazards might be categorized as grave dangers, while others might not. It is unclear, for example, whether temporary infertility would be considered to be a grave danger, even though it could have a permanent effect on an employee's ability to reproduce, particularly if the female of the couple is approaching 40 years of age. In the absence of a grave danger, however, OSHA must promulgate a permanent standard, which may take more than a year to produce, thus allowing some workers to be exposed to the hazard in the interim.

Even where a grave danger exists, the ETS procedure has been held by a Federal appeals court to require an exhaustive statement of reasons, indicating on which data OSHA is relying, why those data are sufficient to show the existence of a grave danger, and why the particular standard is necessary for the protection of employees. Preparing such an exhaustive statement of reasons could be sufficiently time-consuming to render the ETS mechanism ineffective for reproductive health hazards.

OPTION 1:
Congress could maintain the status quo.

This would probably result in OSHA refusal to issue ETSs for hazards that produce certain reproductive health effects (e.g., temporary infertility) that may not be considered grave dangers by the courts. In addition, the requirement of an exhaustive statement of reasons means that ETSs are less likely to be promulgated quickly when a genuine public health emergency occurs.

OPTION 2:
Congress could amend the "grave danger" language of the OSH Act.

This would allow OSHA to respond quickly to public health concerns, including reproductive health hazards, that are not incurable, permanent, or fatal, without fear that a court will require the agency to proceed by way of the cumbersome and time-consuming formal rulemaking process. The disadvantage of this option was recognized by Congress when the grave danger language was adopted. Emergency temporary standards can result in substantial compliance costs to an affected employer, yet they are effective only for 6 months and generally require less supporting evidence than do permanent standards. Congress wanted to spare employers the expense of complying with temporary standards unless a substantial workplace danger warranted the regulation.

OPTION 3:

Congress could amend the OSH Act so that all that is required when an ETS is issued is notice of OSHA's reason for issuing the standard and access to the scientific data on which it relied.

This would allow an ETS to be issued for an agent that is reasonably suspected, though not yet proven, to be hazardous.

Compensation for Job-Induced Reproductive Harm

Even when there is full cooperation among labor, industry, and government, prevention of occupational disease may not always be successful. In some cases, a substance may not be recognized as hazardous until some workers are injured. Even in cases where the hazard is recognized and exposure avoided, accidents occur. A manufacturing or design flaw may make engineering controls or personal protective equipment malfunction. A human error by an employee may result in release of a substance. Exposure to multiple substances both inside and outside the workplace, as well as personal lifestyle and medical factors, may yield unanticipated interactions. All of these scenarios have two things in common: they are *unpredictable* events leading to injury and they will probably continue to occur with uncertain frequency in spite of all preventive efforts.

The issue that remains, therefore, concerns the personal and financial costs of occupational disease in general and reproductive health hazards in particular. While the *personal* cost of reproductive, sexual, or developmental injuries must ultimately be borne by the affected individuals and their families, these individuals may be morally entitled to place some or all of the financial burden on other parties associated with the injurious workplace situation.

Most workers cannot collect compensation for their reproductive injuries. As discussed previously, not only do most workers' compensation systems fail to provide remedies for job-induced reproductive failure, they also deny workers access to court-awarded relief. (Since injured offspring are not covered by workers' compensation statutes, they may press their claims in court.)

Should compensation for a worker's reproductive or procreative injury be provided? If so, should it be provided through court-awarded remedies under State tort law or through workers' compensation schemes, either at the State or Federal level? Since a workers' compensation award is generally the only remedy available to compensate a worker with nonreproductive occupational injuries, it may be rational to extend coverage to job-induced reproductive injuries. Historically, the underlying theory of compensation law is to award benefits only for those injuries that cause a diminution in earning capacity. Workers' compensation can be viewed as being designed to protect the worker from economic insecurity and not as a form of "damages" in the sense of relieving the victim from all of the effects of the injury. Yet the exclusivity rule prevents injured employees from seeking compensatory damages in court, even when the employer is negligent. Since it limits the worker's ability to collect damages, workers' compensation can also be viewed as a form of limited restitution. Because of this conflict, a policy choice is presented in which legislators must weigh the relative interests of the employer, the public, the injured worker, and the integrity of the workers' compensation system.

Several theories underlie the responses of State courts, legislatures, and compensation boards to reproductive harm claims made pursuant to workers' compensation statutes. The narrowest theory is the view that actual wage loss is required for any benefits other than medical. A potentially broader view requires evidence of loss of earning capacity, though not necessarily actual wage loss. The most generous theory, adopted by only a handful of States, claims that the health and functions of the whole man or woman should be used as the standard for measuring the validity of a claim and its compensability. Reproductive or procreative impairment may be covered under such theories because it may have life-shattering effects without negative economic implications.

Virtually all State workers' compensation systems follow one of the two narrower theories, thereby providing a remedy for reproductive injuries only when they affect earning capacity. It is a justifiable option to limit the scope of State compensation plans to the occupationally disabled. But no one claims that it is justifiable to base denial of a tort remedy on the fact that the reproductive injury was job-related if the case falls outside the State system for compensating occupational injuries.

Regardless of whether compensation is provided through the workers' compensation system or the tort system, the problem of assigning moral, legal, and financial responsibility is complicated by uncertainty concerning the relationship between a particular workplace exposure and a particular injury. Scientifically conclusive evidence that a particular workplace exposure caused or contributed to an injury is rare. Test results showing the effects of a substance on animal reproductive or procreative capacity, or on embryo/fetal development, must be interpreted with caution, and research on human exposure presents a number of moral and pragmatic constraints that may confuse the assignment of causation. Furthermore, determination of whether there is a statistically significant relationship between workplace exposure and a medical condition may require study of large numbers of exposed employees; in some cases, the number of workers exposed to the suspected hazard may be smaller than the number of subjects needed for ensuring valid and reliable results. In any event, a court of law or a workers' compensation board may be unwilling to rely solely, or even substantially, on the results of epidemiologic or toxicologic investigations to support claims for compensation.

Given the scientific uncertainty as to causation of most reproductive dysfunction, compensation boards and courts are faced with a choice between compensating too few and compensating too many. If the court or compensation board requires a high degree of scientific certainty, then the tribunal can be relatively certain that it has not paid on fraudulent or erroneous claims, but some genuine cases of job-induced reproductive impairment will go uncompensated due to lack of sufficient proof. If the tribunal accepts less scientific evidence to support claims, fewer meritorious cases will go uncompensated but more erroneous claims will result in a windfall to the claimant. The expense of paying the erroneous claims will fall directly on industry, which funds the workers' compensation program, and ultimately on the consumers of that industry's products. The question therefore arises as to how the burden of scientific uncertainty should be allocated among the various concerned parties.

OPTION 1:

Congress could enact a Federal statute, or State legislatures could add specific provisions to State workers' compensation statutes, to cover loss of reproductive and procreative function even when nondisabling.

Workers' compensation schemes already provide scheduled benefits for some types of injuries in the absence of wage loss (e.g., for loss of an eye, limb, or digit). If coverage for reproductive injuries is adopted, the amount of compensation should be the value that the legislature places on the reproductive impairment; when a worker suffers reproductive or procreative impairment without a wage loss, there is no justification for tying the cash benefit to an existing wage level.

Proposals for occupational disease compensation at the Federal level have generally used job disability or earnings loss as a criterion for compensability. Such legislation would fail to result in compensation for most reproductively injured workers.

OPTION 2:

A Federal statute could be enacted or State legislatures could amend their workers' compensation laws to provide workers with the right to pursue a tort remedy for injuries falling outside the workers' compensation law.

If legislators do not want to extend workers' compensation coverage to nondisabling reproductive injuries, they could adopt this option so that injured workers can sue employers who are allegedly responsible for their injuries.

Adopting this option would probably result in an increase in liability actions. A comparison of the costs of compensating individuals with occupationally caused asbestosis suggests that moving occupational disease cases into the tort system will result in higher awards to injured workers, as well as higher legal expenses, than does placing these cases under the umbrella of workers' compensation. Court proceedings may also take longer than those for workers' compensation; and court proceedings are generally less likely to result in compensation due to the more stringent evidentiary standards that they apply.

OPTION 3:
Reproductive impairment claims could be carefully disaggregated into those suitable for the compensation system and those suitable for the tort system.

This would necessitate variations on the legislative actions suggested above for the first and second options. For example, physical impairment of a worker's reproductive system may be determined to be suitable for the State compensation system (with the necessary amendment and benefits schedule), whereas harms to other members of the worker's family may be determined to be suitable for the tort liability system (as they are at present).

Reducing Uncertainty: Issues in Research

Given the existing level of reproductive dysfunction, it is difficult to know whether the level of risk now tolerated represents the inevitable and irreducible consequence of life in the 20th century, or whether it represents an excessive and reducible risk to the reproductive health of workers and their potential offspring. Additional research on reproductive health hazards can reduce the degree of uncertainty.

From the point of view of workers, increased funding for research is intimately linked to their "right to know" about the substances to which they are exposed. Only informed workers can make informed choices. From the point of view of employers, more research could lead to better understanding of the actions necessary to both protect workers and inform them of potential risks. From the point of view of society, more research could reduce scientific uncertainties and lead to more reasoned consideration of policies to protect the reproductive health of working men and women.

There are practical considerations to be weighed, however. How much research is enough? How should resources be allocated among the various agencies and between basic and applied research? The results of basic research are often not immediately applicable and their impact is difficult to measure. It might be possible to place a monetary value on a new in-vitro assay that reliably and validly tests for specific developmental effects, but how can a monetary value be placed on the prospect of reducing the incidence of spontaneous abortion?

Several types of studies, from research at the molecular level to epidemiological studies on human populations, are necessary to elucidate the causes and consequences of suspected reproductive health hazards. This effort includes basic research to better understand the physiology of reproduction and the mechanisms of action of toxicants. More efficient techniques need to be developed to assay reproductive and developmental effects. Mathematical models for accurately extrapolating dose-response effects from animals to humans are needed. The reproductive endpoints in animals that reliably predict concordant effects in humans need to be clarified. Human populations need to be better monitored and more studies need to be done in the workplace.

The workplace is the laboratory for occupational health research. Occupational health research and monitoring activities are currently carried out by the larger firms, and both toxicology and epidemiology research efforts are sponsored by trade associations. However, some researchers report difficulty in gaining access to industrial settings in order to carry out research on workplace-related health effects. Companies are in a difficult position because they fear liability for injured workers could result from such studies. Congress might limit corporate liability in the case of companies that cooperate with researchers in order to provide an incentive to cooperate. However, this option could place an unnecessary burden on injured workers by denying them full compensation for their injuries.

In a period of budget-tightening, congressional oversight to ensure adequate review of research priorities and scientific standards may be in order. In addition, some measures could improve the quality of data inexpensively. For example, such low-cost options as recording the occupations of both parents on birth records could provide information on whether birth defects are correlated with occupation. Occupational histories of both parents could also be added to the Birth Defects Monitoring Program (CDC survey), and the NCHS National Health and Nutrition Examination Survey (HANES).

Most basic research on human reproductive physiology is carried out in university laboratories sponsored by the National Institutes of Health (NIH) or the National Science Foundation (NSF). Basic research in toxicology is carried out in universities as well as by the National Institute for Environmental Health Sciences (NIEHS), the National Toxicology Program (NTP), EPA, the Food and Drug Administration (FDA), and NIOSH. Work on improved methods of risk assessment, including use of new assays and development of mathematical models for extrapolation from animal data, is being carried out by these same agencies. The Centers for Disease Control is carrying out several surveillance efforts to monitor levels of reproductive impairment in the population. Both EPA and NIOSH are also conducting epidemiology studies. NIOSH can have a positive impact on the quality of epidemiology studies done in industry through its Health Hazard Evaluations. These studies can increase knowledge of human effects, and can be used to further cooperative efforts between government and industry. Congress, through its appropriations and oversight functions, could assign priority to particular types of research and improve its quality.

Chapter 2

Introduction to the Study

CONTENTS

Page
Introduction 31
Prevention of Reproductive Impairment 32
The Population at Risk 33
Historical Perspective 33
Evidence of Reproductive Health Hazards 35
Worker Perception of Risk 36
Risk Assessment and Management of Harmful Agents 37
This Assessment 38
Chapter 2 References 39

Figure

Figure No. *Page*
2-1. Civilian Labor Force Participation Rates for Women 16 Years and Over, Selected Years 34

Chapter 2

Introduction to the Study

INTRODUCTION

Protecting the reproductive health of male and female workers is necessary because reproductive capacity is fundamentally important, both to individuals and to the health of future generations. Because reproductive dysfunction manifests itself in and through a variety of effects, and because these effects are difficult to measure, policymakers may never have complete information regarding the full extent of reproductive health dysfunction. The management of uncertainty, therefore, stands as a central issue in the protection of reproductive health.

This chapter summarizes the nature and complexity of the issues surrounding reproductive health hazards in the workplace, outlining what is known and unknown about agents that may cause harm, the number of people potentially exposed, the nature of research on reproductive hazards, and the risk assessment process in Government regulatory agencies. The historical perspective of women in the workplace is discussed in terms of their changing fertility patterns, and the importance of occupational safety and health measures, worker education, and engineering controls is stressed.

The reproductive system involves many physiological processes, and its functioning is integrated with numerous other organ systems. Reproductive health dysfunction thus has repercussions for general health status. Alterations in sex hormone metabolism or production may, for example, increase the risk of heart disease or certain cancers in men and women. In women, alterations in sex hormone metabolism may cause premature menopause which, in turn, increases their risk for developing osteoporosis. The more immediate effects of reproductive system damage are infertility or subfertility. Reproductive impairment can also affect offspring in various ways.

Hazards to reproductive health include chemicals, drugs, infectious agents, radiation, physical factors, aspects of lifestyle such as the use of tobacco or alcohol, and stress. These hazards may be found virtually anywhere—in the home, in the environment, and in the workplace. This study is confined to reproductive health hazards found in the workplace, where most Americans spend a substantial portion of their lives.

The Federal Government is committed, through legislation, to ensuring as safe and healthy a work environment for its citizens as is administratively and technically feasible. The United States is also committed to a second important social goal, which sometimes appears to conflict with the commitment to protect the health and reproductive capacity of workers and their offspring: equal opportunity for men and women in the workplace. These commitments are complicated by the biological dependency of an embryo/fetus[1] on the pregnant woman. The embryo/fetus, an involuntary presence in the workplace, may need additional protection from exposure to harmful substances beyond that which may be required to protect the health of the worker.

A number of recent events have focused attention on exposure to reproductive health hazards, intensifying public concern over the presence of such hazards both in and out of the workplace:

- **Drug-related damage to children whose mothers ingested apparently harmless drugs during pregnancy.** Use of the nonprescription drug thalidomide by European women to treat minor headaches and insomnia caused major congenital malformations in their children. The thalidomide episode heightened public awareness that a drug can damage the fetus even when it is not harmful to adults.

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[1] Gestation is commonly divided into three stages: 1) the blastocyst, from conception until about week 3; 2) the embryonic, from week 3 to about 8 or 9 weeks; and 3) the fetal, from 8 or 9 weeks until birth. The blastocyst stage is often subsumed within the embryonic stage in order to simplify terminology (see ch. 3).

The use of the prescription drug diethylstilbestrol (DES) by pregnant women in the United States to reduce the risk of miscarriage caused an increased frequency of a rare form of vaginal and cervical cancer in daughters born to these women. Daughters of mothers who took DES are more likely to have structural anomalies in their reproductive organs (6,24,40). Earlier evidence, which had suggested that sons of women who took DES are at higher risk for incidence of structural anomalies in their reproductive organs, has not been confirmed by a recent study (11).

- **Damage to parents and offspring exposed to toxic substances as a result of industrial accidents:** Minamata disease (brain damage resembling that associated with cerebral palsy) in Japan illustrated the potentially devastating effect of industrial pollution on unborn children as well as on adults. In the Japanese city of Minamata, industrial waste containing methyl mercury contaminated the fish eaten by local inhabitants, causing deaths among adults and children, and major congenital defects in children born in the area. More than 10 years elapsed before the cause of the symptoms was officially acknowledged (29).
- **The potential for reproductive damage to adults and their offspring posed by exposure to toxic substances released in industrial accidents:** The escape of a cloud of dioxin from a trichlorophenol plant in Seveso, Italy, and the accidental release of radioactive materials at the Three Mile Island Nuclear Power Plant in Pennsylvania have not, to date, been linked with reproductive damage. They have, nonetheless, served to heighten public awareness of the potential health hazards of industrial processes.

There has also been increased attention given to the effects of such other hazards to reproduction as alcohol consumption, ingestion of illegal drugs, and smoking. These hazardous agents can impair reproductive health and sexual capacity in adults and can have adverse effects on the developing embryo/fetus. They differ, however, in that individuals can control their use and are often aware of the potential health risks posed by use or ingestion of these substances.

PREVENTION OF REPRODUCTIVE IMPAIRMENT

Reduction of preventable reproductive impairment would lessen the need for policies to deal with the consequences of such impairment. A visible, serious, and persistent commitment to safety by both management and labor appears crucial to preventing workplace impairment of reproductive function. Workplace-induced damage to reproductive function can be minimized by such specific measures as reducing exposures through engineering controls (e.g., ventilation), placing physical barriers between the worker and the source of the hazard, substituting nonhazardous materials for hazardous ones, using personal protective equipment, training workers in the safe performance of tasks, initiating repeated, systematic inspections of the workplace for emerging or previously undetected hazards, and rotating jobs or changing tasks to reduce exposure to the hazard. This latter action could, however, have the opposite effect in that greater numbers of workers would be exposed if job rotation were the only means instituted to reduce exposure. Control technologies are extensively described in the recently completed OTA assessment, *Preventing Illness and Injury in the Workplace*, 1985.

It is important to monitor workers for evidence of reproductive health impairment prior to and during workplace exposure, and to adequately compensate those who have been harmed by such exposure. This report assesses current levels of knowledge of the causes of reproductive impairment and detection of such impairment. It also analyzes the regulatory and legal apparatus for reducing exposure to reproductive health hazards and compensating for reproductive impairment when it occurs.

THE POPULATION AT RISK

Ascertaining the extent of exposure to hazards in the workplace is crucial. How many workers are at risk? How many workers are of reproductive age, and how many of these workers are exposed to reproductive hazards? In what occupations are workers more likely to be exposed to reproductive impairment? What is the extent of reproductive dysfunction in the total population?

In 1984, the number of individuals in the American work force totaled 106.3 million, according to the Bureau of Labor Statistics (BLS). Men constituted 56.3 percent (59.8 million), and women, 43.7 percent (46.5 million) of this total. Approximately three-fourths of employed women were of reproductive age (16 to 44).[2] Reproductive age limits for men are more difficult to identify because reproductive function is less strongly correlated with chronological age.

There are no reliable estimates of the number of workers potentially exposed to reproductive or other health hazards at present. The National Institute for Occupational Safety and Health (NIOSH) is, however, now surveying industries for the purpose of obtaining these data, which will be tabulated by sex but not by age. Preliminary information will be available in late 1985 (8,26).

Estimates of the proportion of U.S. women who were employed during their pregnancies indicate that in 1980, 63.2 percent of married women over 20 years of age who had delivered a live infant were employed at some time during the 12 months prior to the birth of their children. Of these women, an estimated 17 percent, or 314,000 mothers, worked in industries and occupations in which they faced possible exposure to 10 potential teratogens (13).[3]

In humans, only one-fourth to one-third of fertilized eggs are likely to survive to term (43). Prior to the third month of pregnancy, about three-fourths of spontaneous abortions show chromosomal or other abnormalities (1,2,12). Some congenital malformation is present in 3 percent of live births in the United States. Some serious developmental defect is diagnosed by the end of the first year in another 3 percent of live births. Although rates of congenital malformation do not appear to be rising, the causes of these malformations are unknown in 60 to 70 percent of these births (10,14).

An estimated 8.4 percent of U.S. couples in which the wife is of childbearing age are infertile[4] (15). In some cases this inability to bear children appears to correct itself; in other cases the infertility persists. The causes of infertility are also unknown in a high proportion of cases.

The rates of such other manifestations of reproductive dysfunction as impotence, contaminated breast milk, or early menopause are unknown. The extent to which the chemical, physical, and biological agents to which individuals may be exposed in the workplace contribute to unexplained impairment of reproductive functioning is also unknown.

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[2] Reproductive age limits for women vary according to the source. Although reproductive biologists usually define reproductive age as from 15 to 44 years, the Bureau of Labor Statistics data cover women only from age 16 onward.

[3] The results of this survey are limited because only married women who delivered a term live birth were included, only three physical agents and seven chemicals were labeled potentially teratogenic, and the exposure of the women in the sample was not measured. Instead, potential exposure was linked to the occupations that women reported. Only nonpharmaceutical, "recognized" animal teratogens were included. Recognized animal teratogens are defined as two positive findings from at least two different laboratories and in at least two different mammalian species.

[4] This figure does not include couples in which one spouse has been surgically sterilized.

HISTORICAL PERSPECTIVE

Interest in protecting reproductive health traditionally has focused on women as bearers of children. One of the earliest references to hazards to women's reproductive health is found in the writings of Aristotle, who observed that "foolish, drunken, and harebrained women most often bring forth children like unto themselves, morose and languid" (7). And in Judges 13:7 of the Old Testament the woman who is to bear Samson is advised, "Behold, thou shalt conceive and bear a son: and drink no wine or strong drink." Only in the last 20 years has the importance of male reproductive health and its contribution to healthy children been widely recognized.

Social concern for hazards to women as bearers of children appears at several points in the history of women in the workplace. This concern has intensified during periods when women entered the workplace in relatively large numbers.

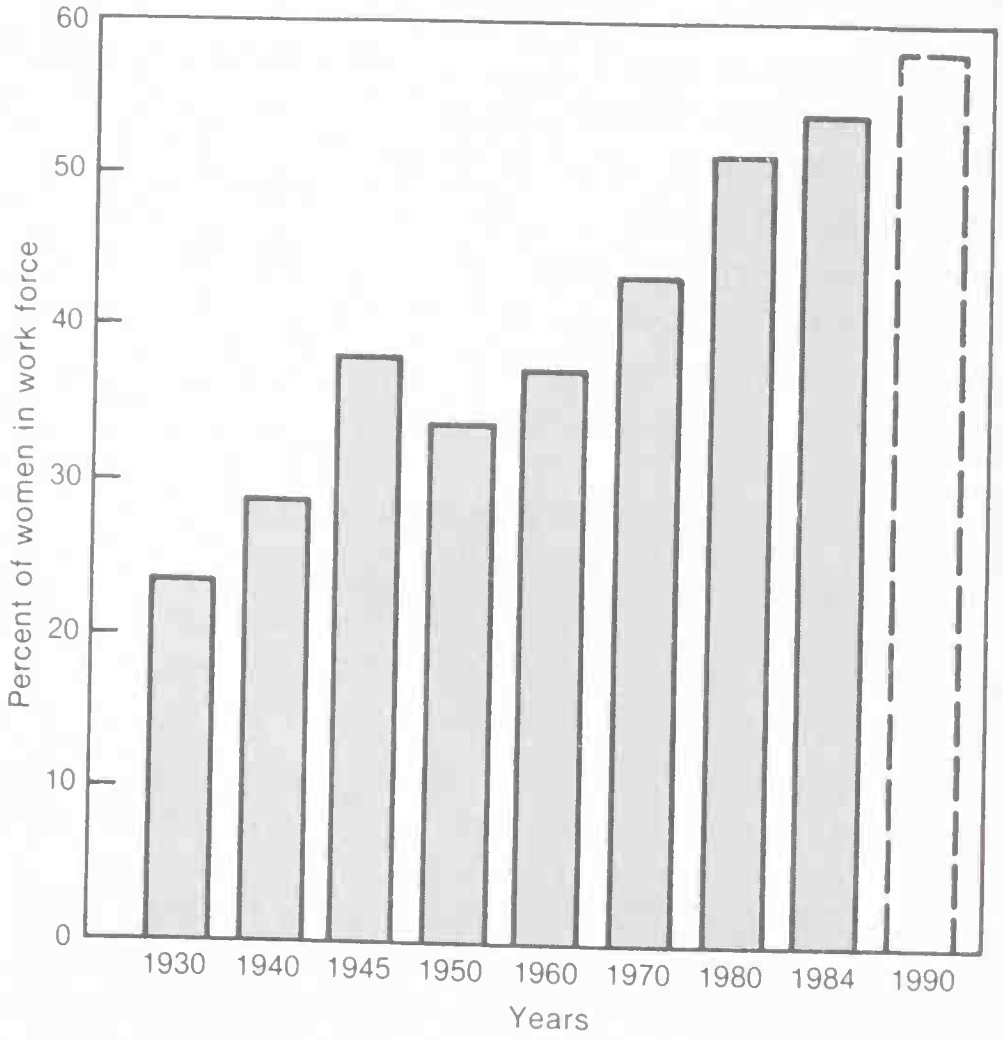
Before the Industrial Revolution women played an acknowledged role in economic life. In agrarian England, male wage earners were paid lower wages because their wives also earned wages. With the eradication of home industries during the Industrial Revolution, women were squeezed out of the economy. During this period the powerful image of woman as preserver of home and hearth flourished, obscuring the role of woman as wage earner. With the emergence of the middle class, a wage earner could make enough money to support a wife, children, and sometimes servants. Women of that era who were not married or who had been widowed had difficulty obtaining jobs that paid well because of the widespread conviction that a woman's place was in the home (28).

The view of women as lifelong homemakers has been perpetuated in the 20th century by the misperception that fewer children and less time-consuming household chores have "pulled" women from the home into the workplace. Smaller family size has not, however, been a decisive factor in the return of women to the workplace. While the birth rate (number of children born annually per 1,000 women of childbearing age) has declined, *more* women today are having at least one child. From 1910 to about 1960, most American women either bore no children or had only one or two children. Until the 1950s, about one in five U.S. women who reached age 35 to 39 had never given birth to a child. Another 20 percent had given birth to only one child. Since the 1960s, the percentage of women who are childless or have only one child has fallen to about 1 in every 10 women of childbearing age.

The persistent image of woman as preserver of the home is also belied by the fact that one-fifth of U.S. women were employed outside the home at the turn of the century (an underestimate because women who labored on farms were undercounted). Before World War II, the proportion of women employed outside the home was nearly 30 percent. This proportion rose to 38 percent during the war, returned to 30 percent immediately thereafter, and has risen steadily since 1945 (28). In 1960, 38 percent of women over 15 years of age were employed; by April of 1984, this percentage had climbed to 54. Some 58 percent of American women are expected to be in the labor force by 1990 (36) (see figure 2-1).

The proportion of married women who are employed has also increased rapidly, from 31 percent in 1960 to 55 percent in 1982. Married women with children accounted for most of this increase. Among married women with children 6 to 17 years of age, the proportion employed rose from 39 percent in 1960 to 62 percent in 1980. Among married mothers with younger children,

Figure 2-1.—Civilian Labor Force Participation Rates for Women 16 Years and Over, Selected Years



SOURCE: U.S. Department of Labor, Bureau of Labor Statistics, *Handbook of Labor Statistics*, Bulletin 2175, December 1983.

the proportion employed more than *doubled*, from 19 percent in 1960 to 45 percent in 1980 (13). By March of 1984, BLS reported that 46.8 percent of married women with children under a year old were in the labor force, compared with only 24 percent in 1970. The sharp rise in numbers and proportion of women workers over the past 10 years has been accompanied by growing concern for their safety. Evidence of the risk to the reproductive capacity and sexual functioning of both men and women posed by toxic exposures has continued to mount during this period.

EVIDENCE OF REPRODUCTIVE HEALTH HAZARDS

The effects of occupationally induced disease on the reproductive system were first described in 1775, when Percivall Pott detected the link between chimney sweeps and scrotal cancer. He observed that scrotal cancer occurred almost exclusively in chimney sweeps and that "the disease in these people seems to derive its origin from a lodgment of soot in the rugae of the scrotum." Pott thus also identified the first known carcinogen. Interestingly, a 1962 report on his work points out that:

> . . . the mechanism of action of soot or its active ingredient is not understood, even after 187 years of enormous technological development, and the easiest, most effective method to control scrotal soot cancer is the same as that available to Percivall Pott and his contemporaries: prevention by avoidance of contact (22).

Physician Alice Hamilton, a pioneer in occupational health, brought the plight of female lead workers to public attention in 1919. Although she also demonstrated evidence of negative health effects in male workers, she was particularly interested in the causes of the more severe effects observed in women. She showed that the adverse health effects in these women and the higher infant mortality among their offspring were due not to their being "the weaker sex" but to the fact that women workers came from economically disadvantaged circumstances. More women than men were suffering from lead poisoning, for example, because men were more likely to be members of strong unions (which gave them some protection from adverse working conditions), were better paid, and had better living conditions. Women were more likely to be young and unmarried or to be widows, since married women were discouraged from working, and were unorganized, underpaid, and poorly housed (9). They came to the workplace undernourished and ill and were further weakened not only by the lead but by the effects of long hours, poor living conditions, and low pay.

To date, most studies of reproductive hazards have been carried out on wives of workers and their offspring or women and their offspring (4,19). The 1977 case involving exposure to 1,2-dibromo-3-chloropropane (DBCP), a known carcinogen, was one of the first to highlight the importance of hazards that affect male reproductive function. Informal discussion among male workers in a California pesticide factory manufacturing DBCP disclosed the fact that their wives had been having trouble conceiving since the husbands began working at the plant. After considerable discussion, one worker convinced five others to submit semen samples for analysis; all samples were determined to be grossly abnormal. All of these men worked with DBCP (41,42). Soon after the discovery of abnormal sperm at this and other plants, the Occupational Safety and Health Administration (OSHA) issued an emergency temporary standard that reduced exposure levels. A final standard was issued in March 1978 (43FR; 11514). DBCP was later banned by the Environmental Protection Agency (EPA) except for specific limited uses (spraying of pineapple plantations in Hawaii). EPA banned all uses in January 1985, and stipulated that existing supplies in Hawaii must be phased out by 1987. A subsequent study (20) indicates that, except in cases of exposure greater than 100 hours, the effects of DBCP on male fertility appear to be reversible. However, there is some evidence of an altered sex ratio in subsequent births to wives of the exposed workers (21) (see chapters 4 and 7).

The policy ramifications of this incident are also significant. Male reproductive capacity was found to be endangered by DBCP, but men of reproductive age were not removed from their jobs. Instead, the hazardous agent was banned. In cases where the potential developmental hazard is paternally mediated, male workers have not been removed. The treatment of women workers in similar circumstances has, in certain cases, been reversed: when developmental hazards to the embryo/fetus have been identified, the women, rather than the hazards, have been removed. In at least two instances female X-ray technicians were removed from their jobs because of suspected risks, and in another case, women had themselves sterilized because they believed it was the only way they could retain their jobs (see chapter 8).

Since the regulation of DBCP in 1978, only two other standards, those for lead and for ethylene oxide, have been developed to protect workers from reproductive health hazards as well as other health hazards. These standards reduce allowable exposure levels and require mandatory posting of signs warning of risks to health and the reproductive system and mandatory employer education of employees with regard to health risks. In the case of ethylene oxide, regular physical examinations with attention to reproductive function are required, and in the case of lead, counseling with a physician is recommended if a pregnancy is planned (49FR 25734; 50FR64; 43FR 52952).

WORKER PERCEPTION OF RISK

Even if all risks could be accurately estimated and all workers fully informed and free to reject risks without other economic or social constraints, workers' actions would still be guided by personal perceptions of risk. The element of risk is a cost that is weighed against other costs and benefits in the personal decisionmaking process. Several features motivate an individual's acceptance of risk (3,5,27):

- the seriousness of the consequences,
- the perceived probability of personal impairment or misfortune,
- the voluntariness of the dangerous activity,
- the familiarity of the risk, and
- the availability/awareness of alternatives.

The inability of an individual to obtain information on which to base a decision is a source of stress. Among the coping mechanisms individuals use when faced with uncertainty is denial. When the safety of an activity is unclear, they may reduce or exaggerate the risk in order to support their choices. Another mechanism is to consider oneself immune from risk: "I am a safe driver; I won't have an accident." Others seek information from external sources, relying on "experts," or the media. A consequence of this tendency is often a distorted sense of the risk inherent in some of the dangers people face. They tend to overestimate the likelihood of highly publicized events while underestimating more common events that elicit less public notice (5,27).

Although there is some evidence that workers mistrust employers, believing that they put profits before safety, evidence from the 1977 Quality of the Workplace Study (23) indicates that 84 percent of the workers questioned believe that their employers do inform and will continue to inform them of any dangerous or unhealthy conditions to which they are exposed on the job. There has been little quantitative analysis of employee risk perception, however. A recent qualitative study (18) describes worker perceptions of risk, fears of being harmed, and perceptions of employer neglect with regard to potential exposure, but provides no representative sampling of worker attitudes.

RISK ASSESSMENT AND MANAGEMENT OF HARMFUL AGENTS

The practices of risk assessment and risk management are changing, as are their underlying concepts. The protection of workers and others from the harmful effects of ionizing radiation emitted at nuclear powerplants was until recently a major focus of concern. As more and more chemicals have been produced, the emphasis of risk assessment and management has turned to the effects of chemicals that may be toxic. Attention has shifted from protecting the human genome from the mutagenic effects of X-rays and radiation to protecting the population from the specific disease effects of often proprietary chemicals produced by individual companies.

Assessing and managing the risks of chemicals and other agents are complex undertakings. Most of the 5 million chemicals now in existence are probably not harmful at typical exposure levels. The National Academy of Sciences (17) estimates that there are about 53,500 chemicals to which individuals in the population potentially could be exposed. This total includes everything from industrial solvents to food additives, however. Many chemicals are manufactured in small quantities or are used in small amounts in research laboratories. Of the more than 48,000 chemicals[5] listed in the Toxic Substances Control Act (TSCA) inventory, only about 12,800 are manufactured in quantities of more than 1 million pounds per year, 13,900 are manufactured in quantities of less than 1 million pounds per year, and 21,700 are produced in unknown amounts (17). It is therefore unlikely that many people will be exposed to more than a few of these chemicals. But because no publicly available toxicity information exists for more than 70 percent of the chemicals included in the TSCA inventory, it is not possible to evaluate their health effects (17). In the case of chemicals for which there is sufficient information to undertake a health hazard assessment, factors such as dose, number of people exposed, conditions of use, and costs of testing must be taken into account in establishing priorities for health hazard evaluation and risk assessment.

The manufacturer is responsible for testing new chemicals when testing is required. Manufacturers must submit a Premanufacture Notification to EPA for substances included under TSCA, for example. But because TSCA requires no standard tests, the data need be only those that the company has available (30,31,32) (see chapter 7). For chemicals in commerce, EPA can issue a rule requiring that certain tests be undertaken by the manufacturer if EPA officials believe that the chemical poses a potential hazard.

In risk assessment, scientists evaluate the risk to find out whether the suspected hazard is real, and if so, the extent of risk to humans from exposure to the hazard (16,39). Scientists use epidemiological and toxicological evidence to predict the health effects of exposure of individuals or populations to hazardous materials and situations. Risk assessment includes: 1) hazard identification, 2) dose-response assessment, 3) exposure assessment, and 4) risk characterization (16; chapter 6):

- **Hazard identification** is the determination of whether a particular agent is or is not causally linked to particular health effects. In order for a substance to be identified as a reproductive or developmental hazard, it must be causally linked to reproductive or developmental impairment.[6]
- **Dose-response assessment** is the determination of the relationship between the dose or magnitude of exposure to an agent and the probability or incidence of the health effects in the population. Estimating human reproductive health effects is difficult because data are most often available only for animals.
- **Exposure assessment** is the determination of the extent of human exposure before or after application of regulatory controls. Exposure can occur in different patterns over time (chronic or acute); it can occur by different routes (inhalation or through the skin); and particular groups of workers may be more likely to be exposed.

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[5] 1982 estimate: this figure now exceeds 63,000.

[6] Developmental toxins may act from the time of conception until puberty, while reproductive toxins may interfere with reproductive or sexual functioning from puberty through adulthood (see ch. 3).

- **Risk characterization** is the description of the nature and often the magnitude of human risk, including attendant uncertainty. All of the issues in the risk assessment process are summarized and evaluated in order to determine the potential risk of the hazard.

Risk management, which follows risk assessment, involves deciding what to do about problems that have been identified in the assessment process. The goal of risk management is to control the risk. Decisionmakers must be able to demonstrate that when a regulation is enacted, there will, for example, be fewer deaths, or less sickness. The policy alternatives are weighed in order to select the most appropriate regulatory action. A host of legal, scientific, economic, and ethical issues attach to risk management (16,38) (see chapters 7 and 11).

Despite a growing body of information concerning the effects of reproductive health hazards and the risks they pose, legislators, regulators, industrial scientists, and managers are confronted by differing levels of uncertainty in efforts to manage potential risks. What is uncertain is likely to differ with each situation. There may be uncertainty as to which agents are harmful because workers are exposed to more than one hazardous agent in the workplace, or there may be synergism among a number of factors (including nonoccupational factors) that cause reproductive impairment. The evidence of toxic effects may come only from animal data, making extrapolation to humans difficult, or there may be a substantial time lag between cause and effect. Decisions regarding the management of reproductive risk must be made within the context of two important Federal statutes:

1. the Government's authority to protect workers, so far as is feasible, from exposure to hazards that could damage their reproductive systems (Occupational Safety and Health Act); and
2. the right of women and men to have equal access to employment opportunities, working conditions, and wages (Title VII of the Civil Rights Act).

The complexity of this decisionmaking is increased by the potential for harm to an embryo/fetus, which can come from either or both parents' exposure to toxic substances in the workplace or from exposure to substances parents may bring home on clothing and equipment.

THIS ASSESSMENT

This study examines the issue of reproductive health hazards in the workplace from three perspectives: scientific, legal, and ethical. Chapter 3 describes the fundamentals of reproductive biology, the mechanisms of action of reproductive and developmental toxins, and reproductive dysfunction in the population as a whole. Chapter 4 presents the scientific evidence for reproductive health hazards in the workplace, including chemical, physical, and biological agents. Chapter 5 reviews technologies for assessing human reproductive function. Chapter 6 describes the nature of the complexities in data collection and evaluation, and discusses the risk assessment process and regulatory agency activities with regard to guideline development for reproductive risk assessment.

The legal issues are discussed in chapters 7 through 10. Chapter 7 covers the prevention of injury; chapters 9 and 10 cover compensation for injury. Chapter 7 analyzes the regulatory process as it affects reproductive risk assessment and regulatory policy in a discussion of activities at OSHA, EPA, and the Nuclear Regulatory Commission (NRC). It also discusses landmark court decisions that bear on the Government's ability to regulate exposure to reproductive health hazards. Chapter 8 continues the discussion of relevant legal issues with an analysis of sex discrimination in employment under Title VII of the Civil Rights Act of 1964, as amended. Chapter 9 deals with workers' compensation systems. Legal liability for causing reproductive damage is assessed in chapter 10, which looks at theories of liability and proof of causation. The issues covered in chapters 9 and 10 are of central importance because of the lack of uniformity in State workers' compensation laws, and the possibility of tort liability of employers if an embryo/fetus is damaged through exposure of the parent to hazards in the workplace.

Chapter 11 is devoted to an analysis of the ethical considerations surrounding the protection of workers and their offspring from reproductive damage.

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Chapter 3

Principles of Reproductive Biology and Development

CONTENTS

Page

Introduction 43

Measurement of Reproductive Function: Relation to Workplace Hazards 44

Normal Reproductive Biology and Development 44
  Hormonal Control Mechanisms 44
  Male Reproductive Function 46
  Female Reproductive Function 48
  Embryogenesis and Fetal Growth 49
  The Pregnant Woman 49
  Coping with Pregnancy Loss 52
  Lactation 53
  Sexual Development: Puberty 53

Abnormal Development 54
  Historical Perspective 54
  Terminology 55
  Mutagens 56
  Impaired Embryogenesis and Fetal Growth 56

Mechanisms of Action of Reproductive and Developmental Toxicants 57

Reproductive Dysfunction in the Population as a Whole 59

Summary and Conclusions 61

Chapter 3 References 62

List of Tables

Table No. *Page*

3-1. Measures of Reproductive Function Readily Obtainable Prior to Fertilization 45
3-2. Measures of Reproductive Function Readily Obtainable After Fertilization 45
3-3. Stages of Embryonic and Fetal Development 51
3-4. Principles of Teratogenesis and Timing of Embryonic and Fetal Toxicity 57

List of Figures

Figure No. *Page*

3-1. The Male Reproductive System 47
3-2. Relation Between Oocyte Number and Age in Women 48
3-3. The Female Reproductive System 50
3-4. Relation Between Age, Oocyte Number, and Menopause 51
3-5. Embryogenesis and Fetal Growth: Three Trimesters of Gestation 52
3-6. The Percentage of Normal Women Who Conceive per Menstrual Cycle and the Outcome of the Pregnancies 53
3-7. Percentage of Surviving and Lost Human Embryos and Fetuses at Different Stages of Pregnancy 54
3-8. Mechanisms of Action of Reproductive Toxins 58

Chapter 3

Principles of Reproductive Biology and Development

INTRODUCTION

Normal reproductive function comes about only as a consequence of interactions among multiple physiological systems. In the narrowest sense, reproduction is the union of sperm and ovum to form a new biological entity. Yet the union of gametes is merely a signal event in the continuum of physiological processes comprising normal reproductive function. Prior to fertilization, for example, the maturation of sperm and egg depends on the coordinated secretion of multiple hormones. At coitus, synchronized neural reflexes and appropriate reproductive behaviors are required to bring gametes together. After conception, embryonic growth depends on the integrity of the zygote and a remodeling of the maternal circulatory system. The later growth and development of the offspring are a function of both prenatal and postnatal nutrition.

For purposes of this report, **reproductive function** is used in the broadest sense possible. It encompasses:

- the functional and structural integrity of the sperm and ova;
- differentiation and development of the internal and external reproductive organs and endocrine glands;
- activation of the adult reproductive system at puberty;
- senescence of the adult reproductive system (e.g., menopause);
- behaviors associated with or subserving reproduction (e.g., libido);
- maternal and paternal prenatal events;
- embryonic and fetal events (e.g., organogenesis);
- maternal postnatal events (e.g., lactation); and
- child health and development.

The significance of some aspects of reproductive function not overtly related to fertility is often underestimated; because they are held to be strictly private matters, many of these subjects tend to go undiscussed. In fact, an individual's reproductive function and, should it occur, reproductive dysfunction, can be of extraordinary personal importance. Impotence, menstrual pain, and loss of libido exemplify instances of reproductive dysfunction that can have substantial impact on individual well-being and human relationships.

Concern about reproductive processes is not limited to the brief periods in an individual's lifetime during which reproduction may actually occur. Reproductive function is an integral part of everyday human health and well-being. Before, during, and after the childbearing years, reproductive hormones may act, for example, on such variables as resistance to heart disease and cancer, immune function, complexion, bone mineral content, and feeling and mood. Threats to reproductive function can take place at nearly any point during an individual's lifespan. In fact, the most insidious hazards to reproductive function may be those whose immediate effects are apparently benign, but whose ill effects surface at a later date.

Viewed from this perspective, the bounds of typical reproductive function and the task of defining atypical reproductive function seem impossibly broad in scope. Yet, by using an array of well-defined endpoints, it is possible to assess human reproductive function in both a qualitative and a quantitative manner.

MEASUREMENT OF REPRODUCTIVE FUNCTION: RELATION TO WORKPLACE HAZARDS

For the couple desiring to reproduce, it may be argued that the only meaningful index of reproductive function is the ability to produce a healthy baby when they wish to do so. At any given time, the couple either can, or cannot, procreate. But, for the purposes of this report, this final common denominator of successful procreation must be dissected into numerous constituent factors in order to: 1) examine the nature of reproductive function and dysfunction, and 2) relate reproductive dysfunction to a potential workplace hazard. Multiple endpoints of reproductive function further serve to define the reproductive status and physiological well-being of the majority of the population who are, at any given time, not procreating.

Endpoints used for measuring reproductive function may be divided into two groups: 1) those serving as indices of reproductive function independent of fertilization, and 2) those serving as such indices after fertilization. There are close parallels between male and female reproductive processes up to the point at which sperm and egg mature. Thereafter, most of the reproductive processes related to procreation occur in the female, as the fetal-placental-maternal system exhibits many stages without counterpart in the male.

Table 3-1 lists measures by which reproductive function may be assessed in adult men and women. The measures listed are limited to those that are readily observable in a relatively noninvasive fashion. In order to have broad applicability in a workplace or outpatient setting, such measures are obtainable by one or more of the following means:

- a detailed patient history,
- a physical examination,
- blood samples,
- semen samples, or
- urine samples.

Table 3-1 illustrates the disparity between the ease with which male and female reproductive parameters can be assessed. That is, **sperm are readily accessible, while eggs are not.** Table 3-2 lists measures by which reproductive function may be assessed in the adult woman and her offspring during pregnancy and after birth. Again, the measures listed are limited to those readily obtainable in the relatively noninvasive fashion just described. A comprehensive discussion of the methods used to assess reproductive function, including more sophisticated methods than those listed in these tables, appears in chapter 5.

NORMAL REPRODUCTIVE BIOLOGY AND DEVELOPMENT

Hormonal Control Mechanisms

In both men and women, the hypothalamus, an area at the base of the brain, serves as a fundamental neural regulator of the body's reproductive function. It receives neural and hormonal input from the brain and endocrine glands and responds to these stimuli by secreting luteinizing hormone-releasing hormone (LHRH) and other hormones. The hypothalamus releases LHRH into tiny blood vessels which surround the pituitary gland. With a target so nearby, LHRH is released in minute amounts and breaks down quickly. As a consequence, this vital reproductive hormone—a telling indicator of reproductive function—is possible but difficult to detect in peripheral blood circulation.

LHRH acts on cells of the anterior pituitary gland to promote secretion of two hormones, luteinizing hormone (LH) and follicle-stimulating hormone (FSH). LH and FSH, known as gonadotropins, direct hormone and gamete production by the testes and ovaries. As the gonads release hormones in response to stimulation by LH and FSH, these gonadal hormones act at the hypothal-

Table 3-1.—Measures of Reproductive Function Readily Obtainable Prior to Fertilization

	Affected individual		
Endpoint	Male	(Both)	Female
Sexual function:	Erection Ejaculation	Libido Behavior	
Endocrine system:		Luteinizing hormone Follicle-stimulating hormone Steroid hormones (androgens, estrogens, and progestins)	Cervical mucus quality
Germ cells:	Sperm number Sperm motility Sperm shape (morphology) Chromosomal integrity Fertilizing ability		
Fecundity:	Testicular integrity Semen quality	Integrity of external genitalia	Ovarian integrity Blockage of oviduct Menstrual regularity Amenorrhea Anovulatory cycles
Secondary sexual characteristics:		Breast development Facial and axillary hair growth Sebaceous glands	
Reproductive lifespan:		Age at puberty	Age at menopause

SOURCE: Office of Technology Assessment.

Table 3-2.—Measures of Reproductive Function Readily Obtainable After Fertilization

	Affected individual		
Endpoint	Female	(Both)	Offspring
Endocrine system:	Human chorionic gonadotropin Steroid hormones, especially progesterone		
Health during pregnancy:	Hemorrhage Toxemia	Fetal death Spontaneous abortion	Morphology Chromosomal aberrations
Perinatal period:		Premature birth Postmature birth:	Death Chromosomal aberrations Birth defects Birth weight Apgar score
Postnatal period:	Lactation		Infant death Childhood morbidity Childhood malignancies Development Behavior
Reproductive lifespan:	Age at menopause		Age at puberty

SOURCE: Office of Technology Assessment.

amus and pituitary gland to reduce the secretion of LH and FSH. In this way, a feedback loop operates, involving the hypothalamus, pituitary gland, and gonads. A defect at any point in the hypothalamic-pituitary-gonadal axis or in the metabolism of their modulator hormones will interrupt the normal pattern of reciprocal hormone secretion among these organs.

The moment-to-moment secretion of LH and FSH is best described as episodic, or pulsatile, with a frequency of 1 to 2 hours under normal conditions. The pattern of episodic gonadotropin secretion represents endocrine signaling from the hypothalamic-pituitary unit to the gonads, thus directing normal ovarian and testicular activity (6,14,58). In addition, larger alterations in the pattern of gonadotropin pulses are correlated with dramatic changes in reproductive function, as in the peripubertal period, at menopause, and in certain pathological conditions. The pattern of hormone secretion is difficult to detect when the plasma concentration of gonadotropins is low, as in prepubertal individuals.

Normal, premenopausal, adult women (but not men) exhibit a second cyclic mode of hormone secretion. This cyclic secretion is marked by a periodic, synchronous burst of LH and FSH release, known as the preovulatory LH surge. Estrogens secreted by the cells in the ovaries act upon the brain to trigger the preovulatory surge. Thus, coordination of both neural and ovarian signals is required for normal ovulation to occur.

In order to map the pattern of LH and FSH secretion—and thus judge hypothalamic-pituitary function—it is necessary to draw serial blood samples at frequent intervals. **A single blood sample yields no information about the pattern of gonadotropin secretion, although it can sometimes identify gross abnormalities in hormone levels.**

The episodic nature of LH and FSH secretion is a consequence of episodic release of LHRH from the hypothalamus. In this way, intrinsic properties of the central nervous system mediate gonadotropin secretion and, ultimately, gonadal function. It is through the central nervous system that psychological, emotional, sensory, and environmental stimuli can profoundly influence reproductive function.

Male Reproductive Function

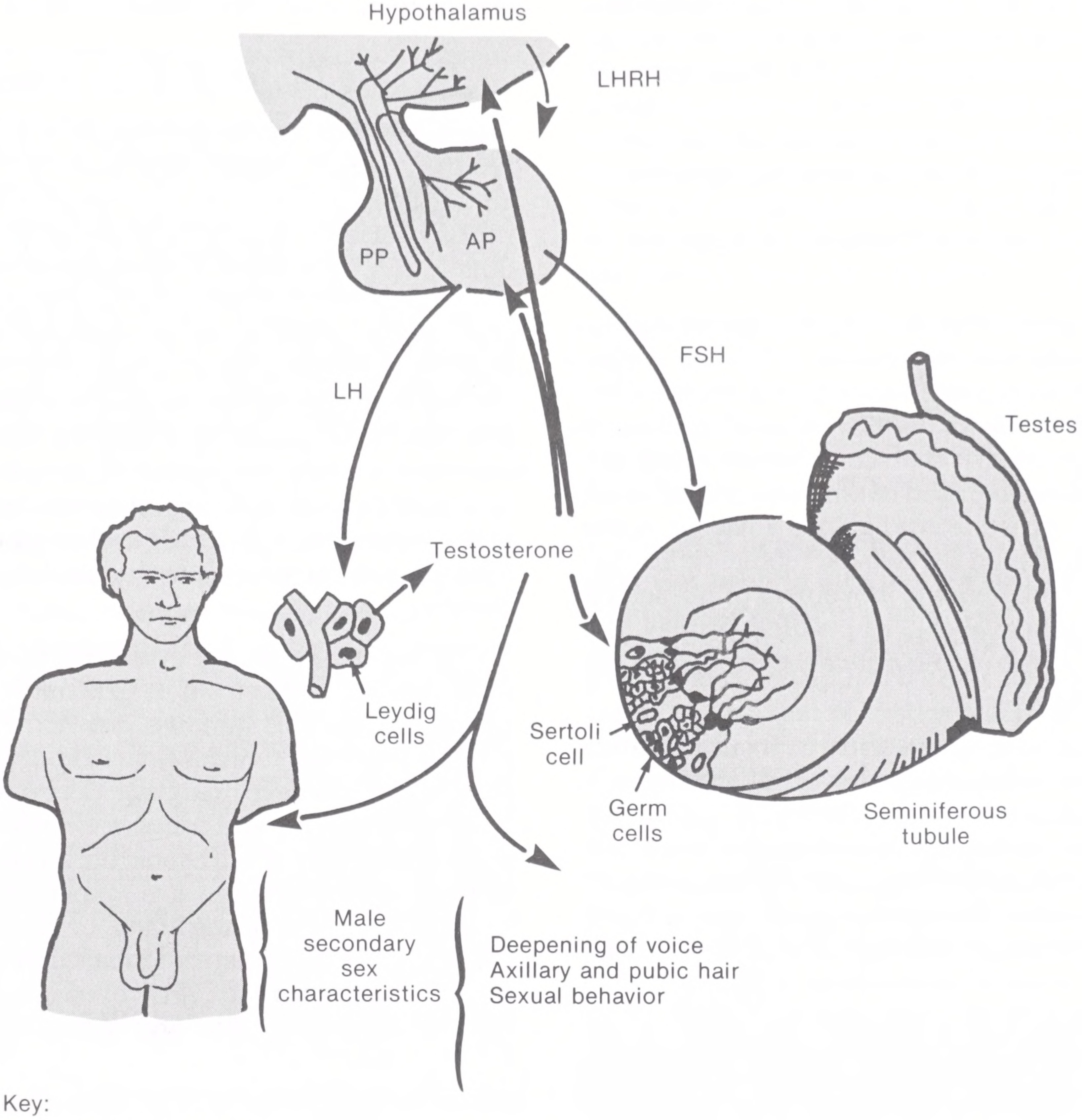
In the male, the testes are the target of the LH and FSH released by the pituitary gland (figure 3-1). The testes serve two functions, producing both gametes (sperm) and hormones, notably testosterone. Sperm develop in the loops of seminiferous tubules within the testes; these tubules make up the bulk of the testes. Testosterone is produced by the Leydig cells, which are scattered throughout the testes and lie outside the seminiferous tubules. Damage to the sperm-producing tubules does not necessarily affect testosterone production by the Leydig cells. However, a deficit in testosterone production by the Leydig cells is likely to be accompanied by impaired sperm production because of feedback to the pituitary and hypothalamus.

Sperm are produced continuously in the testes beginning at puberty and continuing throughout life. A decline in sperm production may occur as men age, becoming apparent in the sixth decade and beyond (22,41). Such an age-related decline in sperm production is not observed in all study populations (44), and the response of the testes to aging is variable (41).

Sperm production begins with division of sperm precursor cells, the spermatogonia, within the seminiferous tubules. Spermatogonia are generally thought of as falling into two broad categories—those in a self-renewing pool and those in a proliferating pool of cells. Most spermatogonia are in the latter. These spermatogonia divide to produce two daughter cells that are destined to become spermatozoa. A few more spermatogonia exist in a pool of cells that renew themselves. These spermatogonia produce two daughter cells that can either remain in the population or commit to the proliferating pool of cells.

When spermatogonia are damaged or killed by a toxic agent (e.g., ionizing radiation) reproductive function in the male may be greatly impaired. There is some evidence that a third type of spermatogonium that rarely divides under normal cir-

Figure 3-1.—The Male Reproductive System


Hypothalamus
LHRH
PP
AP
LH
FSH
Testes
Testosterone
Leydig cells
Sertoli cell
Germ cells
Seminiferous tubule
Male secondary sex characteristics
Deepening of voice
Axillary and pubic hair
Sexual behavior

Key:

PP: posterior pituitary
AP: anterior pituitary
LH: luteinizing hormone
LHRH: luteinizing hormone-releasing hormone
FSH: follicle-stimulating hormone

SOURCE: Adapted from S. M. Harman, "Clinical Aspects of Aging of the Male Reproductive System," *The Aging Reproductive System (Aging, Volume 4)*, E. L. Schneider (ed.) (New York: Raven Press, 1978), pp. 29-58.

cumstances may begin to actively divide to replenish the population of spermatogonial cells, and in this way, the testes may regain sperm-producing capacity. Although it may be temporary, interruption of fertility can have lifelong consequences in that timing of procreation can be crucial. The gonad itself may be the target of toxic agents (e.g., DBCP). In such cases, depending upon the extent of exposure, gonadal damage can be irreversible.

The final stages of sperm maturation take place during passage of the sperm from the testes through the long, coiled epididymis. Maturation involves changes in motility, metabolism, and morphology. Sperm then leave the body in the semen, a fluid comprised of secretions of the seminal vesicles, prostate, and glands adjacent to the urethra. Ejaculation is a two-part spinal reflex that involves: 1) emission, the movement of the semen into the urethra; and 2) ejaculation proper, the propulsion of the semen out of the urethra at the time of orgasm.

The process of forming sperm from primitive stem cells in the seminiferous tubules consumes an estimated 64 to 74 days; the sperm take an additional 9 to 12 days to pass through the epididymis. For this reason, changes in the sperm-producing activities of seminiferous tubules are generally not immediately reflected in ejaculated semen.

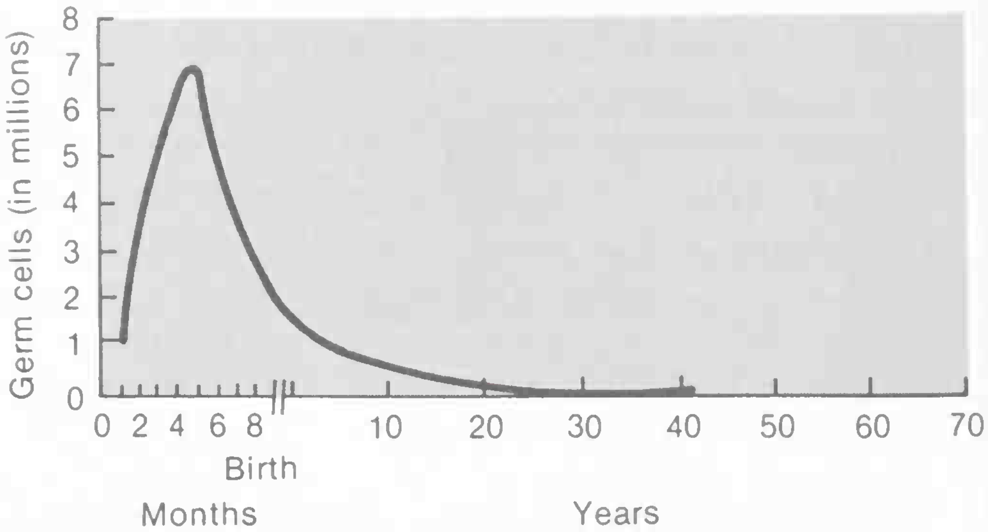
Testosterone has a number of actions. It diffuses into the seminiferous tubules to promote sperm development. Testosterone is also secreted into the general circulation, where it acts at the hypothalamic-pituitary unit to modulate the release of LH. (FSH release by the pituitary gland is modulated by a protein factor called inhibin, which is secreted from the seminiferous tubules.) Testosterone acts to promote growth and development of male sexual organs, causing an increase in size of the penis, prostate, Cowper's gland, and seminal vesicles, and promoting secretory activity of the latter three glands. Male secondary sex characteristics (e.g., increased muscle mass, beard growth, deep voice, and underarm and pubic hair) are all developed and maintained by testosterone. Sex drive in men increases in puberty as testosterone rises, usually decreases in the event of castration, and is restored by exogenous testosterone in men with dysfunctional testes.

Female Reproductive Function

In the female, the target organs of LH and FSH are the ovaries. Within each ovary are primitive germ cells, called oocytes. The number of oocytes in the ovaries is fixed prenatally and is greatest during the fetal stage of development, when it reaches several million. After peaking in the seventh month of gestation, the number of oocytes decreases to fewer than 1 million at birth, and continues to decline markedly throughout life (figure 3-2). **Only about 400 oocytes are actually ovulated during the period of female fertility. In contrast to the continuing renewal of germ cells throughout an adult male's life, no new oocytes are formed after the fetal stage in the female.**

The female menstrual cycle averages 28 to 29 days, but may range from 21 to 50 days (13). Each month, LH and FSH stimulate growth of a selected group of ovarian follicles—small spheres of cells that surround a developing egg. Concomitant with the growth in size and number of follicular cells is the production of estrogenic hormones by these ovarian cells. Estrogens are responsible for the thickening of the uterine lining, or endometrium. Estrogens also stimulate and maintain secondary sex characteristics (e.g., growth of breasts, development of a flared pelvis, and distribution of body

Figure 3-2.—Relation Between Oocyte Number and Age in Women



SOURCE: Adapted from D. R. Mattison, M. S. Nightingale, and K. Shiromizu, "Effects of Toxic Substances on Female Reproduction," *Environ. Health Perspect.* 48:43-52, 1983.

fat to hips and thighs), and induce cyclical alterations in cervical mucus.

Follicular growth continues throughout the follicular phase of the menstrual cycle. One dominant follicle then prevails, while the 20 or more other follicles at the same stage of development begin to degenerate. At ovulation, the dominant follicle ruptures in response to a surge of LH and FSH, and the ovum travels down the oviduct to the uterus. Fertilization of the ovum by a sperm usually takes place in the oviduct, within 24 to 36 hours after ovulation. The follicular cells of the dominant follicle remaining in the ovary form a temporary endocrine organ called the corpus luteum.

During the second half of the menstrual cycle, the luteal phase, the corpus luteum produces high levels of progesterone in addition to estrogens. These hormonal changes prepare the uterus for a possible pregnancy. If a fertilized egg does not reach the uterus and begin to implant, the corpus luteum regresses, the uterine lining is discharged, and menstruation occurs. (Figure 3-3 summarizes the female reproductive cycle.) The luteal phase usually consumes about 14 days. Variability in the length of the overall menstrual cycle, from 21 to 50 days, typically results from varying duration of the follicular phase, rarely from variations in the luteal phase, although shortening of the luteal phase may profoundly affect the ability to support implantation of the fertilized egg (see chapter 5).

Menopause, the cessation of menstrual cyclicity, occurs when the ovary is virtually depleted of oocytes, and is marked by diminished production of ovarian estrogens, bursts of LHRH release, sudden body-temperature fluctuations, and other changes of a longer term. It occurs, on average, at about age 50 (figure 3-4). The destruction of oocytes at any time from the fetal period through adulthood may lead to premature ovarian failure, and premature menopause. As oocytes age, the chances of developmental abnormalities in offspring increase.

Embryogenesis and Fetal Growth

If fertilization of the ovum occurs (24 to 36 hours after ovulation), cell division is initiated and continues during the next 3 to 4 days as the early embryo, called a blastocyst, passes down the oviduct. The blastocyst implants in the lining of the uterus 6 to 7 days after ovulation. During the second and third weeks following conception, extraembryonic membranes are laid down and the development of the three layers of cells (endoderm, mesoderm, and ectoderm) occurs. Thus, by the time the first menstrual period is missed, the embryo is in the primitive "streak" stage.

The embryonic period takes place between weeks 3 and 8 to 9 of pregnancy.[1] This is a critical phase of development, during which cell differentiation proceeds at an accelerated pace. During this period, the brain, eyes, heart, upper and lower limbs, and other organs are formed.

The fetal period is considered to have begun after the major organs have developed. It extends from approximately 8 or 9 weeks of gestational age until birth. This period is both a time of fetal growth and continued biochemical and physiological maturation of tissues and organs. Early in the fetal period, during weeks 9 to 11, the external genitalia differentiate. The growth and development of the nervous system occurs largely in the later fetal stages, during the second and third trimesters of pregnancy. It is important to note that the growth of nerve cells, or neurons, and the formation of connections between neurons, called synapses, continue in humans even after birth. Table 3-3 summarizes the timing of embryonic and fetal development, and figure 3-5 places the periods of embryogenesis, organ-system development, and fetal growth in the perspective of a full-term pregnancy.

The Pregnant Woman

If a fertilized egg reaches the uterus and begins to implant, the nascent placenta produces the hormone hCG, human chorionic gonadotropin. This hormone signals the corpus luteum to continue producing progesterone and estrogens in order to maintain the uterine endometrial lining.

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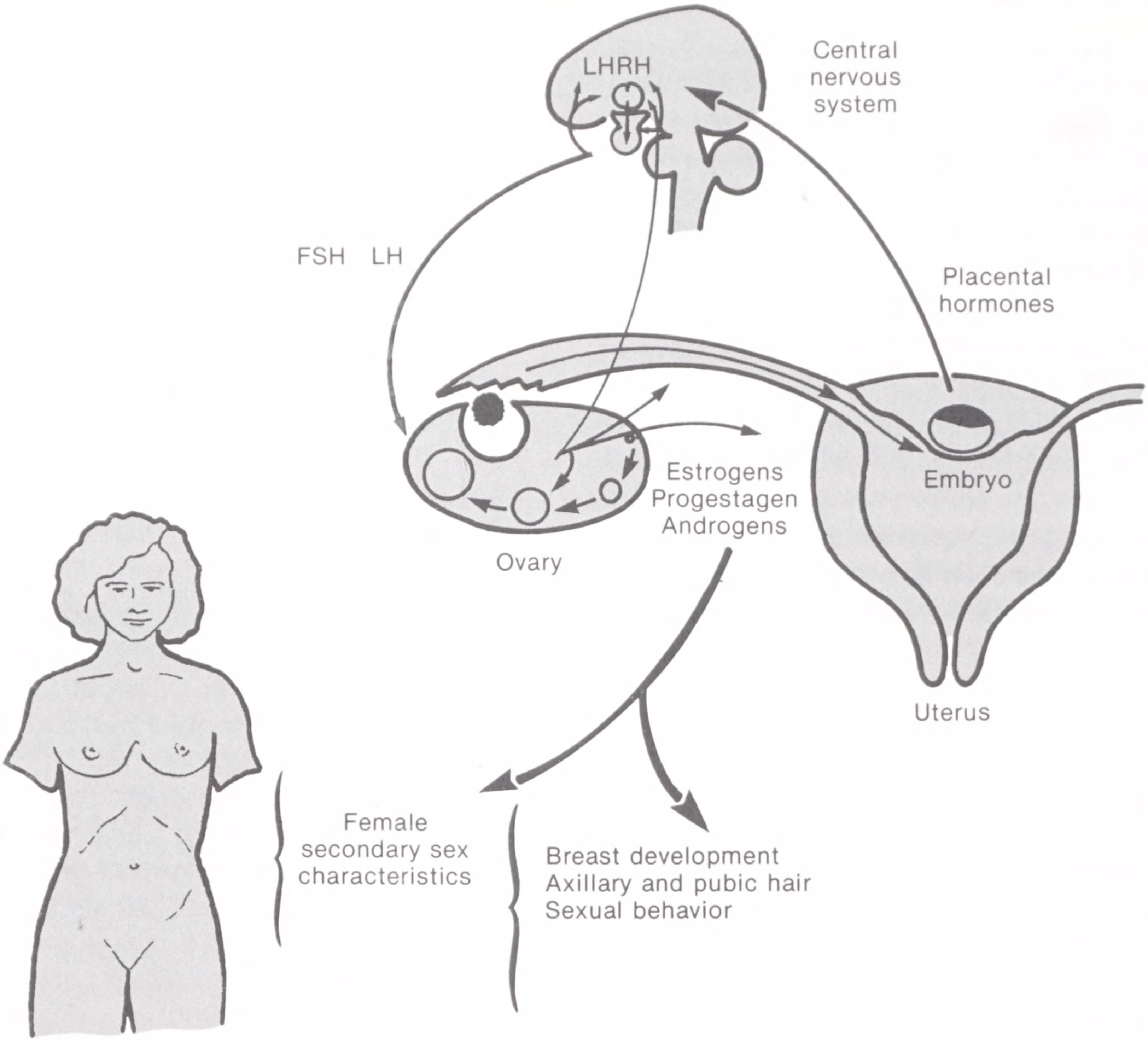
[1] References to time during pregnancy are often made in two ways. If the time from conception, or time of gestation, is enumerated (as in this text), a full term pregnancy spans about 38 weeks. If pregnancy is timed from the last menstrual period, about 2 weeks are added, making a term pregnancy equal to about 40 weeks.

Figure 3-3.—The Female Reproductive System


LHRH
Central nervous system
FSH LH
Placental hormones
Embryo
Estrogens
Progestagen
Androgens
Ovary
Uterus
Female secondary sex characteristics
Breast development
Axillary and pubic hair
Sexual behavior

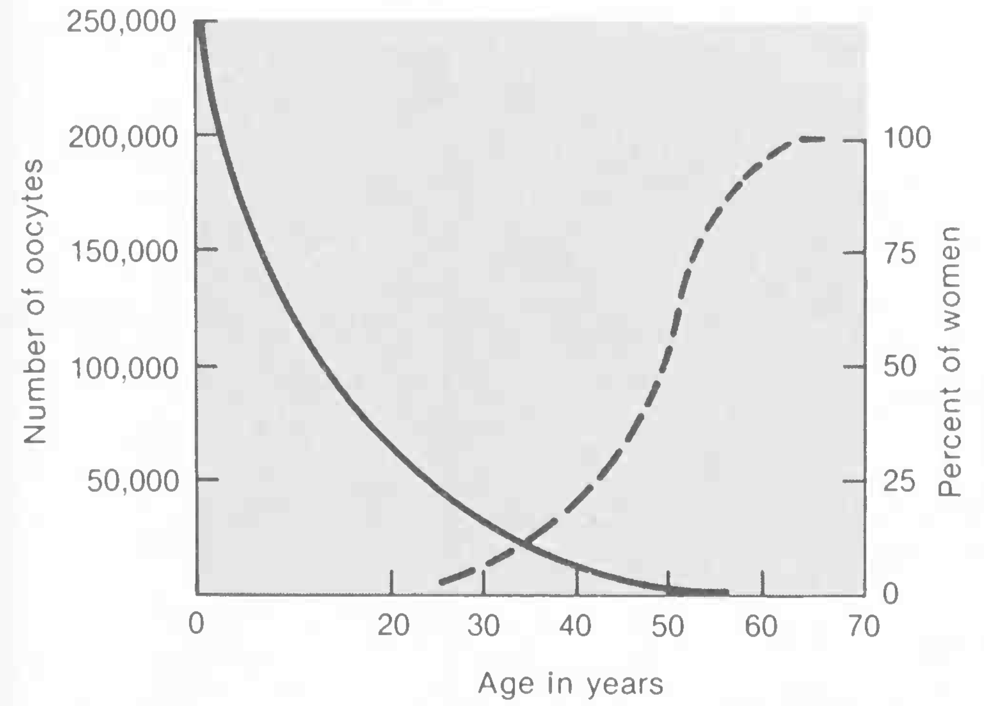
Key:
LH: luteinizing hormone
FSH: follicle-stimulating hormone
LHRH: luteinizing hormone-releasing hormone

SOURCE: Adapted from E. K. Silbergeld and D. Mattison, personal communication, 1984.

Secretion of hCG is the earliest biochemical change indicative of pregnancy. Chorionic gonadotropin has been detected in plasma and urine as early as 6 to 9 days after conception; that is, very soon after implantation of the primitive embryo into the uterine endometrium. Under in vitro conditions, hCG secretion has been detected at 7 days after fertilization, in the absence of implantation (15), suggesting that hCG release by the developing embryo occurs even prior to implantation. In a spectacular demonstration of the diagnostic value of hCG measurement, doubly elevated hCG levels in blood have been used to diagnose the occurrence of twins, just 2 to 3 weeks after conception (23).

During the first 60 days of gestation, the secretion of hCG doubles approximately every 2 days (5). This leads to an exponential rise in maternal plasma hCG concentration with very little individual variation. Maternal plasma hCG levels during the first 60 days of pregnancy can thus be

Figure 3-4.—Relation Between Age, Oocyte Number, and Menopause



SOURCE: Adapted from D. R. Mattison, M. S. Nightingale, and K. Shiromizu, "Effects of Toxic Substances on Female Reproduction," *Environ. Health Perspect.* 48:43-52, 1983.

used to accurately predict gestational age. After 60 days' gestation, hCG levels vary widely and are of little value for predicting gestational age (28,29).

A high rate of embryonic loss occurs during the early phase of the normal reproductive process. It was suggested more than 60 years ago that embryonic death is so widespread in mammals, including humans, that it should be accepted as a normal phenomenon (47). For example, the conception rate per menstrual cycle for a normal couple of reproductive age having unprotected intercourse is nearly 50 percent, whereas the viable pregnancy rate is approximately 25 percent (52) (see figure 3-6). This loss of embryos is particularly high in the very early stages of pregnancy, 1 to 2 weeks after conception. Estimates of embryonic and fetal wastage in women are depicted in figure 3-7. These data have been used to estimate the probabilities of conception, recognizable pregnancy, and live birth in women who are attempting to reproduce. Upon exposure to spermatozoa, the probability of fertilization of an ovum is estimated to be 84 out of 100. By the time pregnancy is recognizable, half of all embryos have been lost. During the remainder of pregnancy, another 25 percent perish and are spontaneously aborted. The entire process—from exposure of an ovum to a spermatozoan through parturition—results in an estimated probability of a live birth of only 31 out of 100 (3). Employing a different frame of reference, the success rate of pregnancies following implantation of the conceptus is estimated to be 57 percent, with 43 percent ending in spontaneous abortion (32).

Pregnancy generates changes in the physiology of the pregnant woman (reviewed in (10)). Her blood volume increases to 150 percent of its nonpregnant volume. The resulting moderate dilution of red cells in the plasma is the anemia of pregnancy and is normal. However, the pregnant woman may be particularly vulnerable to other factors that induce further anemia, including poor nutrition and iron deficiency. Because of the increase in blood volume, her heart works harder, and more blood goes to all her organs.

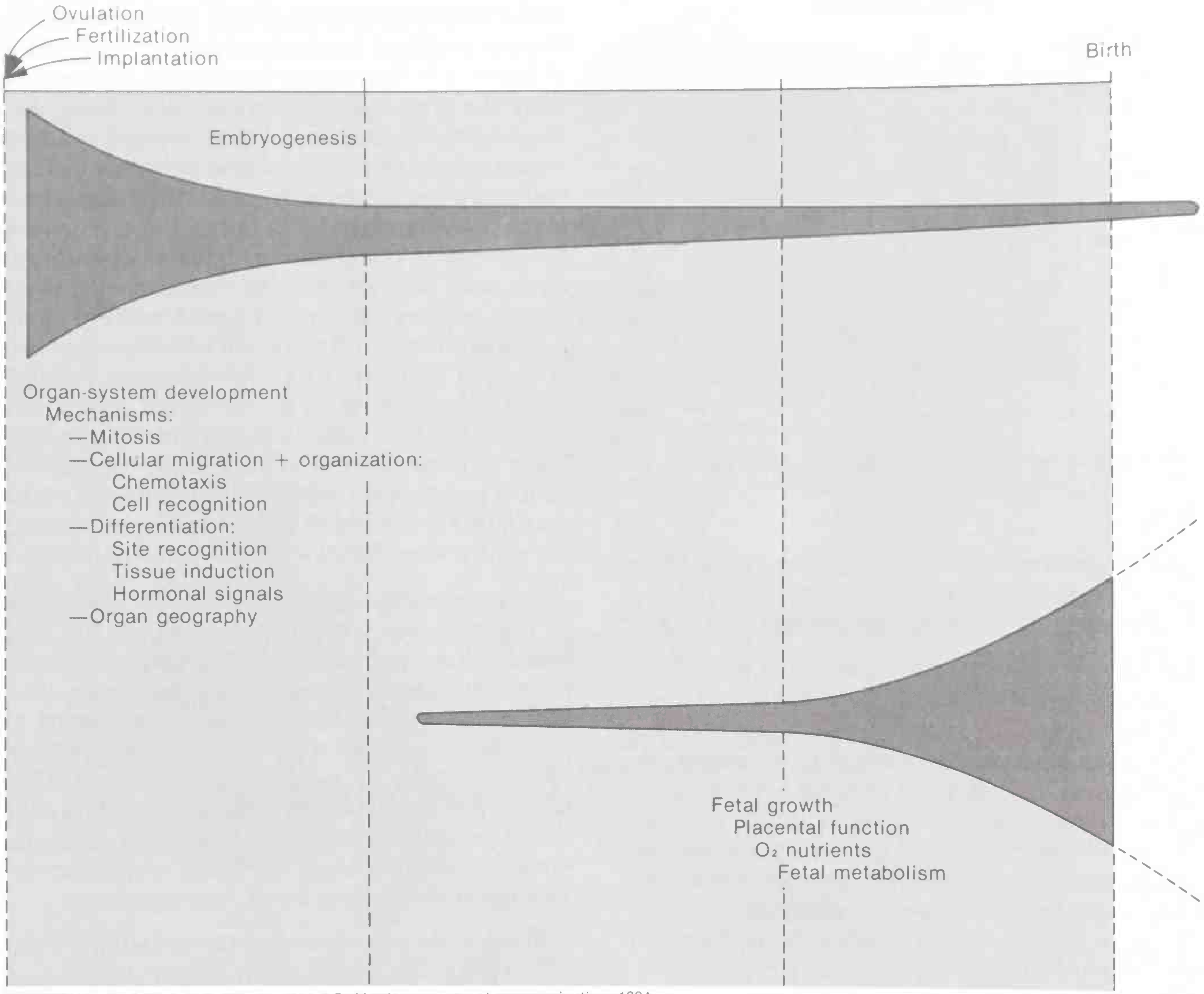
Greater blood volume and the growing weight of the pregnant uterus act in concert to increase

Table 3-3.—Stages of Embryonic and Fetal Development

Period	Time after conception	Stage	Time after conception
Fertilized ovum	First week	Cleavage	1-3 days
		Blastocyst	4-5 days
		Implantation	7 days
Embryonic streak . . .	2-3 weeks	Gastrula	7-8 days
		Neurula	20 days
Embryo	3-8 weeks	Tail-bud embryo	29 days
		Complete embryo	35-37 days
		Metamorphosing embryo . .	38-56 days
Fetus	9-40 weeks	First fetal	56-70 days
		Second fetal	70-140 days
		Third fetal	140-280 days

SOURCE: Adapted from R. H. Blank, *Redefining Human Life: Reproductive Technologies and Social Policy* (Boulder, CO: Westview Press, 1984).

Figure 3-5.—Embryogenesis and Fetal Growth: Three Trimesters of Gestation


Ovulation
Fertilization
Implantation
Birth
Embryogenesis
Organ-system development
Mechanisms:
—Mitosis
—Cellular migration + organization:
Chemotaxis
Cell recognition
—Differentiation:
Site recognition
Tissue induction
Hormonal signals
—Organ geography
Fetal growth
Placental function
O₂ nutrients
Fetal metabolism

SOURCE: Adapted from E. K. Silbergeld and D. Mattison, personal communication, 1984.

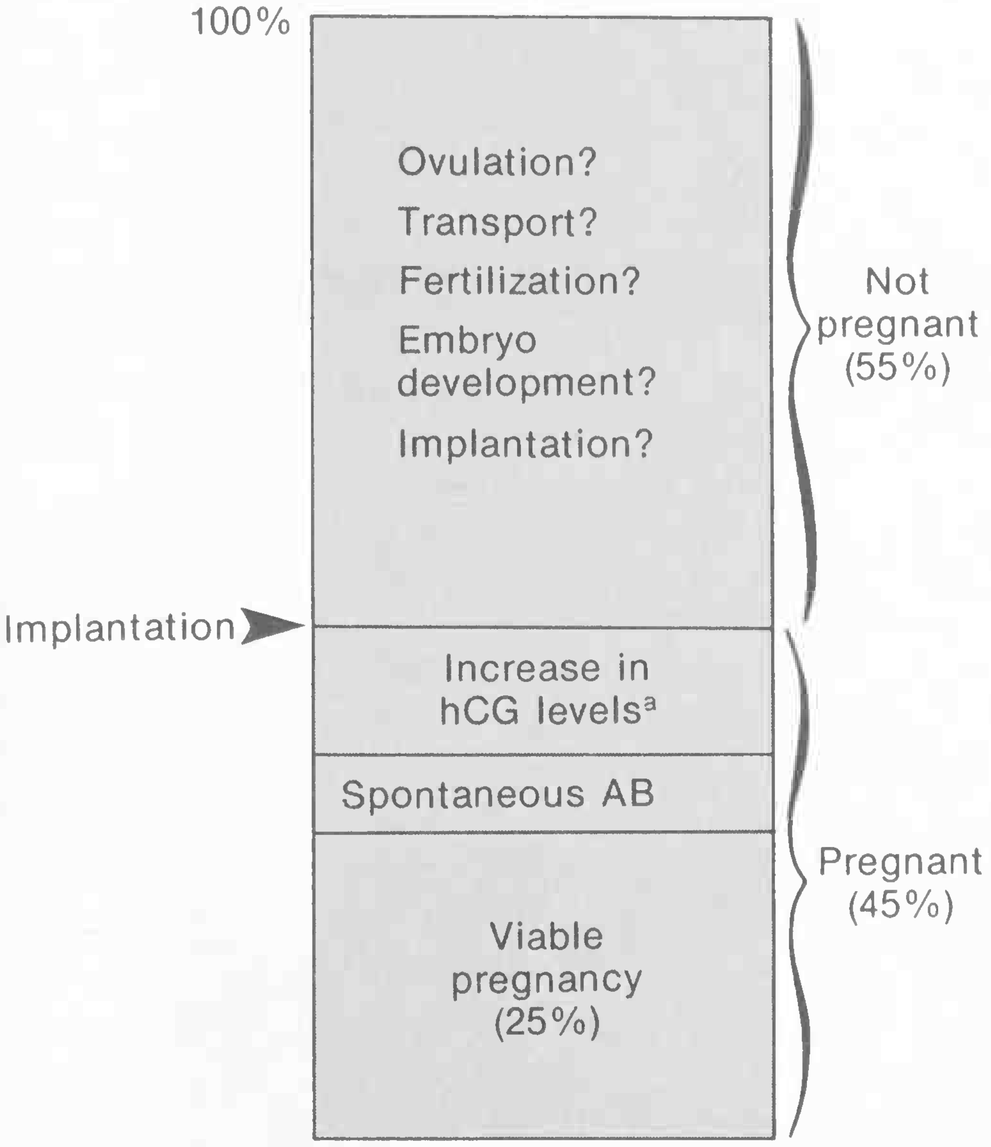
pressure on the leg veins during pregnancy. Sitting or standing in one position may become uncomfortable, and the risk of developing varicose veins in the legs is increased. The weight of the enlarging uterus also increases strain on the lower back. The pregnant woman's kidneys serve to filter wastes from both her blood and that of the fetus. The increased blood flow to the kidneys and pressure on the bladder can cause the pregnant woman to urinate more frequently, particularly as pregnancy progresses.

Coping With Pregnancy Loss

Embryonic or fetal loss causes maternal and paternal grief reactions. The grief pattern seen parallels that which has been described in facing death in adulthood (25), namely:

- shock,
- disorganization,
- volatile emotions,
- guilt,
- loss,

Figure 3-6.—The Percentage of Normal Women Who Conceive per Menstrual Cycle and the Outcome of These Pregnancies



[a] In some pregnancies clinical diagnosis is not made but the woman does have a transient increase in serum human chorionic gonadotropin (hCG) levels.

SOURCE: Adapted from M. R. Soules, "The In Vitro Fertilization Pregnancy Rate: Let's Be Honest With One Another," *Fertility & Sterility* 43(4):511-513, 1985.

- relief, and
- reestablishment of an emotional balance.

A 1984 study found that the strongest stage of grief in pregnancy loss was guilt. This stage took the longest time to begin to resolve, and was the one in which the couples needed the most support and assistance. Women stated that if only they had not jogged, or had sexual intercourse, or fallen, or if they had eaten better, the spontaneous abortion might not have happened. Others had to deal with previous events that represented higher risks, such as medical illnesses or heavy cigarette smoking (30).

Although society is sensitive toward the couple who experiences pregnancy loss, there is a tendency not to express this sympathy. There are, for example, no accepted rituals for mourning an early pregnancy loss. Wakes and funerals are uncommon for a nonviable fetus. In-depth, emotionally supportive counseling sessions are considered an essential part of care for couples who experience a pregnancy loss (30).

Lactation

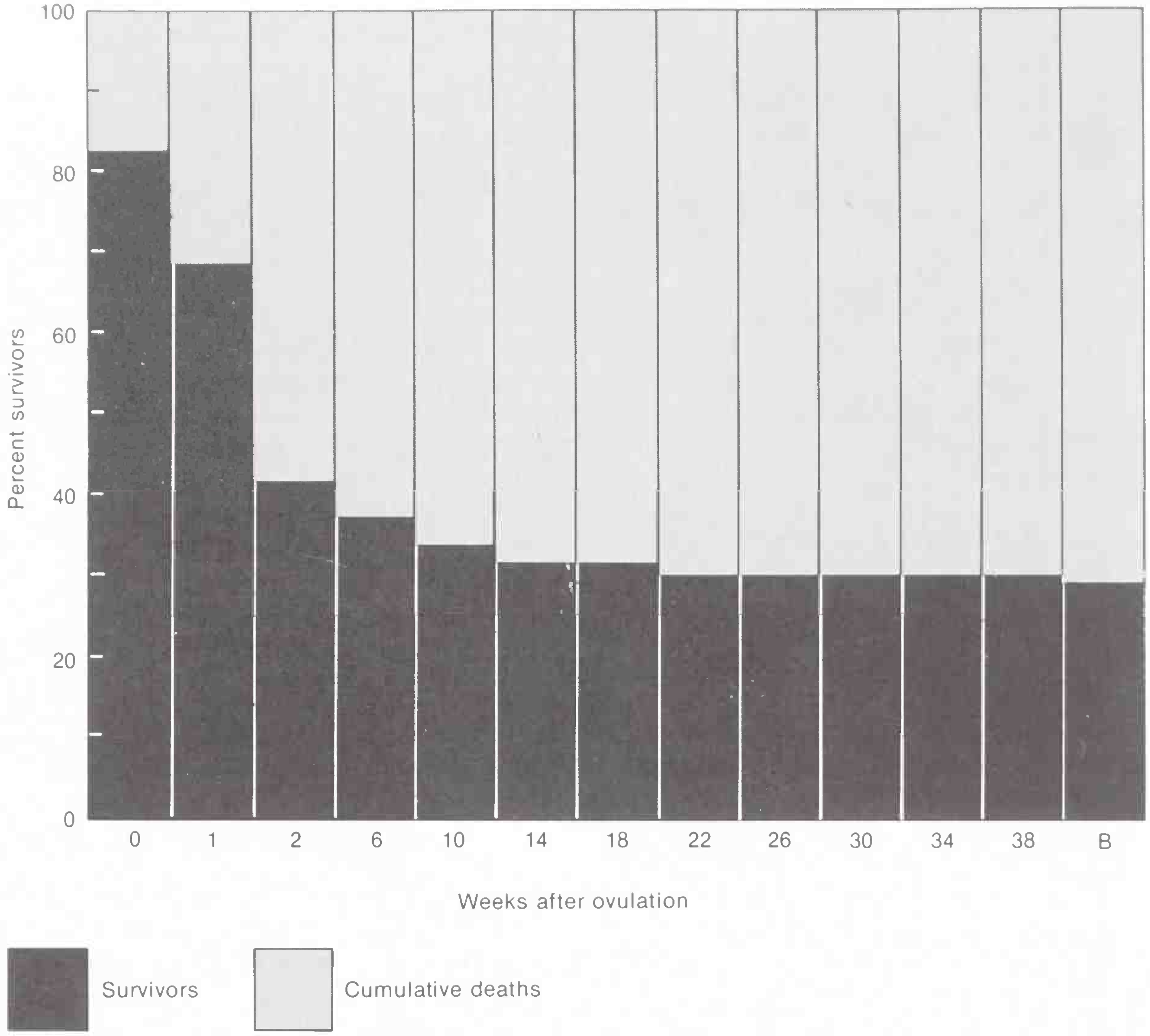
The breast is a complex organ that both synthesizes and excretes. When feeding a growing infant, the mother typically produces a liter of milk per day, containing protein, fat, carbohydrate, minerals, vitamins, hormones, and antibodies. All nutrient components are fully digestible. The product is delivered sterile, on demand, and with the carbohydrate and protein suspended in a mineral/aqueous system. The fat is excreted as a milk-fat globule. Because breast milk is a mixture of both water and fat, it can serve as a vehicle for a wide variety of substances present in maternal tissue or blood. Many constituents present in maternal blood plasma may be present in breast milk. Chemical or drug excretion into breast milk may be accomplished by binding to milk protein or to the surface of milk fat globules. It is also possible that fat-soluble chemicals (e.g., DDT, PCB, most insecticides) may be trapped entirely within the milk-fat globule (2,19,61).

Sexual Development: Puberty

Puberty is the period of transition between the juvenile state and adulthood. During this stage of development, secondary sex characteristics appear and mature, the adolescent growth spurt occurs, profound psychologic effects are observed, and fertility is achieved. These changes are in part a consequence of maturation of the hypothalamic-pituitary-gonadotropin unit, stimulation of the sex organs, and secretion of sex steroid hormones (17). A complex biological and maturational event, puberty actually spans several years, and is not well understood in terms of its onset.

Most American girls (98.8 percent) enter puberty between age 8 and age 13, with a mean age of 11 years (43). They complete their secondary sexual development in an average of 4.2 years, with a range of 1.5 to 6 years (32). Menarche (the first menstrual period) occurs fairly late in the maturational process and is the salient event for the pubertal girl. The first menstrual period appears at an average age of 12.8 years (56).

Figure 3-7.—Percentage of Surviving and Lost Human Embryos and Fetuses at Different Stages of Pregnancy



SOURCE: J. D. Biggers, "In Vitro Fertilization and Embryo Transfer in Human Beings," *New Engl. J. Med.* 304(6):336-342, 1981.

Some sign of puberty is first shown by 98.8 percent of normal American boys between 9 and 14 years, with a mean age of 11.6 years (43). Boys complete secondary sexual development in an average of 3.5 years, with a range of 2 to 4.5 years (33).

ABNORMAL DEVELOPMENT[2]

Historical Perspective

Since the 1950s, tests of the effects of selected chemicals on reproduction have been conducted by the pharmaceutical industry, using animal models. The prospective sires and dams are usually exposed to the test chemical by diet, and measurements are made of reproductive endpoints (e.g., pregnancy rate; successful parturition; number, viability, and growth rate of offspring).

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[2] This section reviews abnormal development of the embryo/fetus; a discussion of abnormal reproductive function from puberty through adulthood appears in ch. 5.

As a consequence of the thalidomide tragedy in the early 1960s (see chapter 2), intensive efforts were mounted to detect substances capable of producing structural abnormalities in developing fetuses. The ability to detect skeletal and external malformations was emphasized, because techniques were available to detect those types of effects (60). These efforts were placed in a practical context as awareness grew that nearly all substances or agents are capable of adversely affecting the conceptus, if the dose is sufficiently great (24).

Methodologic advances since the 1960s have permitted detection of soft-tissue deficits and some functional deficits. These include alterations in central nervous system function (7), intestinal function (11), and respiratory function (42). As a result, the concept of teratology has evolved into a broad concept that includes structural and functional aspects of reproductive and developmental capability.

Terminology

The field of developmental toxicology is evolving rapidly, and its vocabulary is consequently in a state of flux. In late 1984, the Environmental Protection Agency (57) summarized the relevant terminology as follows:

- **Developmental toxicity** is the induction of adverse effects on development occurring up to the time of puberty. The four principal manifestations of developmental toxicity are: 1) death of the conceptus, 2) structural abnormality, 3) altered growth, and 4) functional deficiency.
- **Embryotoxicity** and **fetotoxicity** refer to any toxic effect on the conceptus occurring as a result of prenatal exposure. The distinguishing feature between the terms is the period during which the insult occurs. These terms include malformation, altered growth, and in-utero death.
  - —**Altered growth** is a significant alteration in fetal or neonatal organ or body weight. A change in body weight may or may not be accompanied by a change in skeletal maturation. Altered growth can be induced at any stage of development, may be reversible, or may result in a permanent change.
  - —**Functional teratogenesis** refers to alterations or delays in the postnatal abilities of the individual or organ system, following exposure to an agent during critical periods of prenatal or postnatal development.
  - —A **malformation** is defined as a permanent structural deviation that is generally incompatible with or severely detrimental to normal postnatal survival or development. These types of defects are also called **teratogenic** effects. A **variation** is defined as a divergence beyond the usual range of structural constitution, but which may not have as severe an effect as a malformation on survival or health. Distinguishing between malformations and variations is difficult, since there exists a continuum of responses from the normal to the extreme deviant. Other terminology that is often used, but no better defined, includes **anomaly, deformation,** and **aberration.**

Developmental toxicants thus induce functional teratogenesis, structural malformations, altered growth, or variations. Toxicants can act during either the embryonic or fetal periods, and can kill the embryo or fetus. Developmental toxicants may be equally toxic to both the parents and the embryo/fetus. If exposure occurs at, or sufficiently near to, the adult toxic dose, both the embryo/fetus and pregnant woman are likely to be harmed[3] (21,27).

A teratogen can be defined in several ways. As indicated, the EPA defines teratogenic effects as functional alterations or delays in postnatal abilities and structural malformations that are generally incompatible with or severely detrimental to normal postnatal survival or development. A teratogen can also be defined as a substance that adversely affects the embryo at doses below those necessary to produce overt signs of toxicity in the pregnant woman (53). Yet another definition states that a teratogen is an agent that produces a malformation at any dose (21).

Thalidomide remains the premier, but not sole, example of a chemical—a pharmacologic in this instance—uniquely hazardous to the developing embryo. It has a marked selectivity for a particular target in humans, the limb buds of the conceptus. Thalidomide is able to injure the conceptus at dose levels so small as to be essentially harmless to the pregnant woman. However, most developmental toxicants can affect the woman as well.

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[3] Some substances may be equally toxic to woman and embryo. If exposure occurs at, or sufficiently near to, the adult toxic dose, both the embryo and woman will be affected. The woman may recover, but the embryo can be irrevocably damaged (19).

The evolution of the concept of developmental toxicity and teratogenicity over the past 20 years has implications for public policy. For example, the Toxic Substances Control Act (TSCA; Public Law 94-469), written in 1976, classifies some chemicals as "teratogens," thereby implying the exclusion of substances that may cause other developmental effects. Section 4(b) of TSCA states that testing standards may be prescribed for carcinogenesis, mutagenesis, and teratogenesis by the Administrator of the Environmental Protection Agency. Section 4(e) requires the Administrator to develop a list of chemicals for priority attention. The chemicals listed are those known or suspected to cause or contribute to cancer, gene mutation, or birth defects. Section 10(c) requires coordination between the Administrator and the Secretary of the Department of Health and Human Services for research on rapid screening techniques for carcinogenic, mutagenic, and teratogenic effects of chemicals.

The wording of these sections of TSCA is generally consistent with contemporary understanding of cancer and mutations. However, insertion of the words "developmental toxicants" would clarify the existing statute with regard to contemporary understanding of the word "teratogen."

Mutagens

A mutagen is an agent capable of altering the structure of deoxyribonucleic acid (DNA), the genetic material of a cell. The basic process of mutagenesis may be spontaneous or induced by some agent, and may involve the alteration of a single cell. If the event occurs in a sperm progenitor or egg cell, the cell may die or the mutation may be transmitted to progeny of the affected parent. This kind of mutation, called a germ cell mutation, may be expressed, for example, as fetal wastage, sterility, structural or functional defect, or inherited disease. If the event occurs in a cell other than a sperm or an egg, the result may be cell death or the formation of daughter cells that produce altered gene products or tumors. This type of mutation is called a somatic cell mutation (46). Mutations in somatic cells imply the existence of a germ cell genetic hazard if the inducing agent also reaches the gonads. Mutations may or may not be harmful either to the affected individual or to the progeny.

Impaired Embryogenesis and Fetal Growth

During its earliest phase, prior to implantation and beginning organogenesis, the fertilized ovum (table 3-3) is largely resistant to certain types of toxicants. That is, toxic insults occurring during the preimplantation stages that do not kill the embryo usually do not have an adverse outcome. During this early embryonic period—the first 3 weeks of pregnancy—the most probable effects of toxic influences on the embryo are severe damage and death, followed by spontaneous abortion (16).

After implantation, the organs develop rapidly in a complex series of overlapping and interdependent events. The embryonic period is the primary, although not the sole, period for the induction of congenital malformations. During embryogenesis, the rate of cell division and the timed differentiation of primordial cells into organ systems confer a period of increased vulnerability to toxic effects. This is the period during which most structural teratogens act; functional teratogens may act later on, as well. The expression of teratogenicity varies with dose and with timing of exposure during gestation (51).

During the fetal stages and extending into early postnatal life, major functional and tissue maturation occurs. An agent acting during this period of time can markedly disrupt these processes. Such insults would be expressed not as major gross anatomical abnormalities, but rather as decrements of anticipated function (21). For this reason, most damage occurring in fetal stages is likely to be regarded as a type of functional injury, rather than as the gross malformations or developmental disruptions that may occur during the earlier embryonic period (16).

The major organs are already formed by the beginning of the fetal stages, after which it is too late to cause gross morphological abnormalities. For example, after the palatine shelves have already fused with one another to form the palate, cleft palate cannot be induced by any agent. Nevertheless, a substantial amount of development continues after the embryonic stages, and in-utero exposure of the fetus has been established as capable of producing altered postnatal functional capabilities. Such alterations have been produced in numerous organ systems (e.g., central nervous system, gastrointestinal tract, and cardiovascular system) (21).

Exposure of the developing nervous system to toxic influences may result in enduring behavioral deficits or abnormalities. Behavioral teratogenesis may thus be induced during organogenesis, in the later fetal stages of pregnancy, and even post-partum. Ingestion of mercury, alcohol, or addicting drugs, for example, can cause behavioral deficits or abnormalities in later fetal stages.

The exact nature and severity of induced impairments to embryogenesis and fetal growth depend on such factors as the time of exposure, the severity of exposure, and the nature of the substance itself (see table 3-4). Although it is generally not possible to examine a defective newborn and determine precisely when, during pregnancy, a malformation occurred, it is often possible to determine a gestational age beyond which it could not have been precipitated (21).

Table 3-4.—Principles of Teratogenesis and Timing of Embryonic and Fetal Toxicity

- Teratogens often adversely affect only a portion of exposed individuals; large individual differences in susceptibility exist.
- Susceptibility to embryotoxins depends on the genetic makeup of the embryo and the environmental conditions and lifestyle variables surrounding the parents.
- Toxic agents may be devastating to the embryo but harmless to the parents.
- A toxic agent may produce defects at different levels of biological organization resulting in biochemical, physiological, or behavioral anomalies that may not be apparent at birth.
- A toxic agent may affect the embryo even when given prior to conception either to the mother or to the father.
- The kind of effect a genetic or environmental toxin produces depends on the stage of development during which it acts.
- The same toxic agent may disrupt the developmental program and produce a congenital malformation at one stage, but merely injure an organ or produce no effect at all at another stage.
- The earlier in the formation of a structure a toxic agent acts, the more complete is the damage to that structure.

SOURCE: Adapted from A. S. Goldman, "Critical Periods of Prenatal Toxic Insults," *Drug and Chemical Risks to the Fetus and Newborn*, R. H. Schwartz and S. J. Yaffe (eds.) (New York: Alan R. Liss, Inc., 1980).

MECHANISMS OF ACTION OF REPRODUCTIVE AND DEVELOPMENTAL TOXICANTS

The mechanisms of reproductive and developmental toxicity can be reduced ultimately to some effect that interrupts the normal functioning of a cell, tissue, organ, or organism (8). A toxicant, whether a chemical, physical, or biological agent (see chapter 4), acts by interrupting biological processes, including the transfer of energy and information necessary for normal reproductive function and development.

Following exposure, for example, to a toxic chemical, the compound must be distributed to the target organ (e.g., hypothalamus, pituitary gland, gonad, uterus, epididymis, or liver), where it exerts its toxic effect. Within the target organ, the toxin interacts with a critical cell or subcellular component, disrupting an event necessary for normal reproductive function. If this interaction goes unrepaired, the toxic effect—altered reproductive function—will be produced. The toxic effect may be highly specific and affect only a single function of a single cell type. Or it may be broad and nonspecific, with multiple sites of toxicity within the organism. Within each target, this multistep process precedes the occurrence of reproductive toxicity (34).

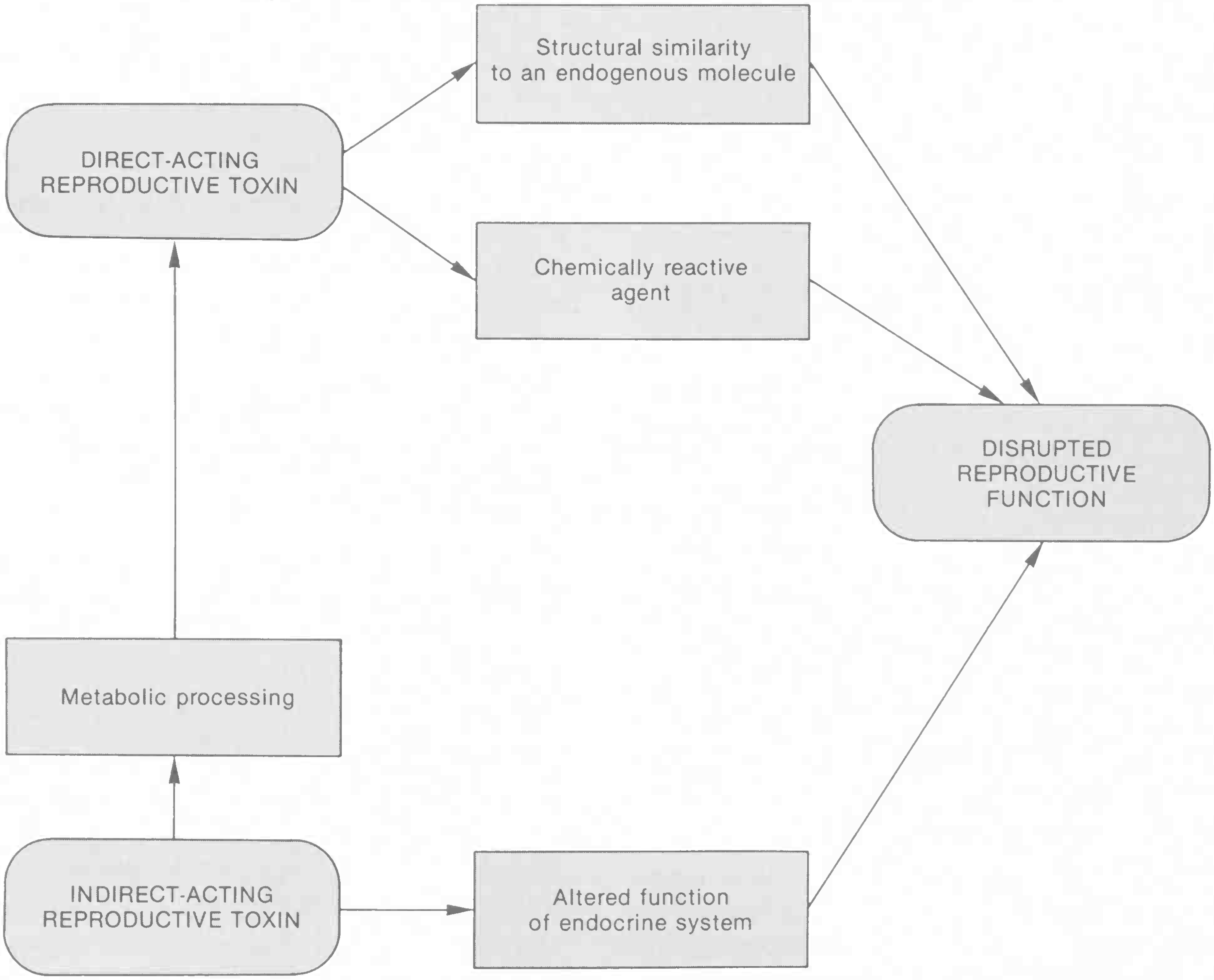
Metabolism of the chemical by the liver or kidneys, for example, may result in toxicity that is more or less apparent. In some cases, a compound may be metabolized and cleared from the body, and no adverse effect will occur. In other cases, metabolic products may be more toxic or long-lived than the original toxin.

Reproductive toxins may act directly: 1) by virtue of structural similarity to an endogenous compound (e.g., hormone or nutrient); or 2) because of chemical reactivity, such as the ability to alter the structure of, or denature, a protein hormone. Some reproductive toxins may act indirectly, requiring metabolic processing or conversion within the body before exerting a toxic effect. The metabolite formed may then act through one of the direct mechanisms of reproductive toxicity (i.e., structural similarity or chemical reactivity). Other indirect-acting reproductive toxins may exert their effects by producing alterations in the body's physiological control systems (e.g., activation or inhibition of enzymes) (34). Figure 3-8 illustrates these mechanisms of action of reproductive toxins.

It is also possible for reproductive toxins to exert adverse effects through multiple mechanisms. For example, polychlorinated or polybrominated biphenyls (PCBs, PBBs) may act indirectly by activation of subcellular enzymes. These same compounds may also act directly by virtue of their ability to mimic the structure and function of steroid hormone molecules (34).

A great deal of attention is being given to research efforts to discover the mechanisms of action of agents known to disrupt development. Current knowledge, however, falls markedly short of identifying even the developmental se-

Figure 3-8.—Mechanisms of Action of Reproductive Toxins


Structural similarity to an endogenous molecule
DIRECT-ACTING REPRODUCTIVE TOXIN
Chemically reactive agent
DISRUPTED REPRODUCTIVE FUNCTION
Metabolic processing
INDIRECT-ACTING REPRODUCTIVE TOXIN
Altered function of endocrine system

SOURCE: D. R. Mattison, "The Mechanisms of Action of Reproductive Toxins," *Am. J. Industr. Med.* 4:65-79, 1983.

quences leading to some adverse effects, much less the precise cellular and molecular mechanisms involved in disruptions of normal structure and function of either the reproductive system or in-utero development (21). Nevertheless, it is possible to enumerate general developmental mechanisms that can be disrupted and lead to altered development. These include:

- faulty cell or tissue differentiation;
- excessive, or in some cases inadequate, cell death during development;
- improper cellular migration;
- faulty intercellular communication; and
- disrupted metabolism, manifested as altered respiration, absorption, excretion, or secretion.

Three issues are central to understanding the mechanisms of action of reproductive and developmental toxicants; these issues also illustrate the overall complexity of reproductive toxicology (34). They are:

- **Species differences:** Differences in reproductive toxicology among species are a reflection of variations among species. In mechanisms of hormonal control, for example, there are differences in anatomy, metabolism, and pharmacokinetics.[4] In some instances, these species differences are poorly understood. A reproductive toxin in one species may not be toxic in another (including humans) because of differences in reproductive or toxicological mechanisms. The teratogenicity of thalidomide is an instructive example of species susceptibility in that rat and mouse are relatively insensitive, while rabbit, human, and nonhuman primates are sensitive (49). Another example is the difference exhibited by rats and mice in sensitivity to oocyte destruction by aromatic hydrocarbons (e.g., benzo(a)pyrene) (36).
- **Gender differences:** This issue is crucial because of the differences in anatomy and biological control mechanisms for reproduction in the male and female. Because of the ease of accessibility of gametes and gonads in the male, more suspect compounds have been screened in animal studies and demonstrated toxic to males than to females. Whether this represents an actual gender difference in gametic or gonadal toxicity or is simply an artifact of experimental designs is as yet unknown. More parameters are accessible for evaluating sperm, for example, than more-difficult-to-obtain oocytes (table 3-1).
- **Time frame for toxicity:** Knowledge of the window of sensitivity during which a structure or function may be affected by reproductive and developmental toxicants is of critical importance. A developing organ such as the ovary (35) may be susceptible to the harmful effects of a reproductive toxin, yet the same agent may have no effect on the developed organ. Little is known, for example, about differences between the immature oocyte and the mature, preovulatory oocyte with respect to susceptibility to reproductive toxins.

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[4] Pharmacokinetics refers to the study of the action of a chemical in the body over a period of time. It includes the processes of absorption, distribution, localization in tissues, transformation into other chemicals with biological activity, and excretion.

REPRODUCTIVE DYSFUNCTION IN THE POPULATION AS A WHOLE[5]

In 1982, approximately 2.4 million married American couples, or 8.4 percent of those in which the wives were of childbearing age (15 to 44) were unintentionally infertile. The epidemiologic profile of infertile couples reveals: 1) a greater proportion of infertile couples among blacks than whites, 2) a tendency to have experienced one or no live births, and 3) a tendency for the woman to be age 30 or over with less than a high school education. Although the overall infertility rate among married couples (excluding those who have been surgically sterilized) has not changed since the 1960s, subgroups of couples in which the wife is age 20 to 24 or black have experienced substantial increases in infertility

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[5] This section is a summary of the detailed analysis of reproductive impairment in the general population that appears in app. A.

(39,40). It is important to note that many infertile couples are only temporarily affected and may eventually bear a viable infant irrespective of medical treatment (12).

The causes of infertility are often complex, difficult to pinpoint, and variable among individuals. Infertility is attributed in roughly equal proportions to men and women among married couples (18). The known and suspected causal factors of infertility can be categorized as:

- **environmental,** including pollutants;
- **pathological,** including infectious diseases;
- **heritable,** such as genetic syndromes;
- **iatrogenic, or medication-induced,** including contraceptive and therapeutic drugs;
- **nutritional;**
- **ascribed,** including race, maternal or paternal age; and
- **sociobehavioral,** including "recreational" drugs, stress, and exercise.

Analysis of these factors reveals large gaps in scientific knowledge of the causes of infertility, and even sparser knowledge about possible synergism with occupational factors.

Infant mortality rates in the United States are higher than those of many developed countries. The proportion of infant deaths due to birth defects has risen to more than 20 percent, because: 1) the rate of birth defects has not fallen as rapidly as the overall infant death rate, and 2) improvements in prenatal and postnatal care have reduced the infant death toll from other causes. The overall infant death rate for blacks is almost twice that for whites, and more than three times higher for infant deaths that are due specifically to low birth weight or prematurity. Although the overall rate of birth defects is lower among blacks than whites, the proportion of black infants of low birth weight is almost twice that of white infants, probably because of: 1) the higher proportion of preterm black infants, and 2) the higher proportion of black mothers possessing risk factors for bearing low birth-weight infants.

Birth defects afflict about 7 percent of live-born infants in the United States (31). About one-half of these birth defects are apparent at birth; the remainder become clinically apparent within 1 year. Some of the most common defects involve the cardiovascular system and the male urogenital system. Many of the more common birth defects, such as Down syndrome or neural tube defects, have a substantial impact on the individual, family, and society because of the severity of their physiological and functional effects. Single neural tube defects (those with no major associated defects) decrease in incidence following a gradient across the United States from East to West and are most common in white and female newborns (26). Several other defects, including Down syndrome and clubfoot, are most common in the Northeast.

The causes of the majority of birth defects are unknown. Individuals may be affected differently by a given causal agent, and some may not be affected at all. Age, health, and personal habits of both male and female, and extent of prenatal care in the female are some of the characteristics that can influence the risk of adverse fetal effects. Attempts to isolate and identify work-related reproductive hazards must take these variables into account (50). The timing and extent of fetal exposure to the agent during gestation may also vary its effect.

Sociobehavioral factors have received much attention in the quest to understand the causes of birth defects. Alcohol is teratogenic when consumed by the mother in large amounts (defined variably) and can result in "fetal alcohol syndrome," characterized by central nervous system dysfunction, mental retardation, growth deficiency, and facial deformities (54). Among neonates of alcoholic mothers, 83.3 percent had birth weights under the tenth percentile compared with 2.3 percent in a nonalcoholic sample (55). In a prospective study of the relationship between birth weight and alcohol consumption during the first trimester of pregnancy in 31,604 pregnancies, the authors found that consuming at least one to two drinks daily was associated with a significantly increased risk of producing a growth-retarded infant. Conversely, consuming less than one drink daily had minimal to no effects on intrauterine growth and birth weight. The authors note that "an occasional drink has only a trivial effect on intrauterine growth" (38). Conclusions regarding the effects of alcohol consumption, although probably valid for heavy drinkers, may be tentative because of the difficulty of assessing all possible impacts on prenatal development. These include factors often associated with excessive alcohol consumption such as smoking, heavy coffee consumption, abuse of drugs, lower socioeconomic status, and poor nutrition. In addition, most studies do not control for the father's consumption of alcohol or other paternal risk factors.

Cigarette smoke and nicotine are also harmful, carrying an increased risk of: 1) prematurity; 2) low birth weight, due partly to fetal malnutrition resulting from depression of placental circulation or maternal appetite; and 3) perinatal death (45,54). A pregnant woman who smokes two packs of cigarettes a day may reduce the oxygen supply to her fetus by 25 percent (1). Effective October 1985, new warning statements were required (Public Law 98-474) on the packages and advertising of all cigarette brands sold in the United States (59). Two of these statements call specific attention to the hazards imposed by maternal smoking upon the offspring, for example:

> **SURGEON GENERAL'S WARNING:** Smoking by Pregnant Women May Result in Fetal Injury, Premature Birth, and Low Birth Weight.

Data on the effects of passive smoking—inhalation of the spouse's or co-worker's smoke by the pregnant woman—on the fetus are not available.

In sum, more complete knowledge of causal factors for both male and female infertility and birth defects in the population at large is needed to accurately isolate and identify reproductive hazards specific to the workplace. Epidemiological surveillance using incidence data is capable of detecting only unusually high rates of infertility or birth defects in certain worker populations, and only after many people have been affected. Even then, epidemiological data are often not sensitive enough to pick up more subtle changes (see chapter 5), and national prevalence data may not pinpoint locally high rates of infertility and birth defects. Furthermore, many indicators of reproductive impairment, such as early spontaneous abortion, are difficult to detect and are therefore underreported.

SUMMARY AND CONCLUSIONS

The complexity of the continuum called reproductive biology and development is masked by a tendency to focus on discrete components of the process, such as the sperm cell or the egg cell or the embryo. Reproductive function also encompasses pregnancy, lactation, child health and development, puberty, adult behavior, reproductive senescence, and the integration of reproductive physiology with the overall health of the individual. Failure to recognize the integral role of each of these components as part of reproductive function leads to an underestimation of the sensitivity of normal reproductive biology and development to perturbation.

Reproductive function in adult men and women can be assessed by relatively simple means, including a detailed patient history, a physical examination, blood samples, semen samples, and urine samples. When only these means are employed, a disparity exists between the ease with which male and female reproductive parameters can be assessed. Sperm are readily accessible, while eggs are not. However, evaluation of the causes of particular aspects of reproductive dysfunction is difficult. Diagnostic techniques are discussed in chapter 5.

Embryonic loss is a normal part of the reproductive process. Only one-quarter to one-third of all embryos conceived develop to become liveborn infants. The remainder are lost at some stage between fertilization and the end of pregnancy. Data such as these are hard to obtain, and estimates vary, because the loss of embryos is particularly high in the early stages, before clinical diagnosis of pregnancy is made.

The terminology of the evolving field of developmental toxicology is rapidly changing. The four principal manifestations of developmental toxicity are: 1) death of the conceptus, 2) structural abnormality, 3) altered growth, and 4) functional deficiency. Structural abnormalities and alterations or delays in postnatal abilities are teratogenic effects. Insertion of the term "developmental toxicant" for the term "teratogen" in the language of TSCA would clarify the existing statute to coincide with contemporary understanding of the word "teratogen."

The complexity of reproduction and development is mirrored by the complexity of biological mechanisms underlying toxicology, which involve absorption, distribution within the body, metabolism (toxification and/or detoxification), excretion, and repair (34).

Toxicants may produce their adverse reproductive or developmental effects by one of several mechanisms. Some agents may act directly, either by virtue of direct chemical action, or by structural similarity to endogenous molecules (e.g., hormone mimics or antagonists). Other agents interrupt reproductive processes indirectly, either by metabolic processing to a direct-acting toxicant (e.g., metabolic activation to form an active chemical), or by altering the normal endocrine balance (e.g., increased steroid hormone clearance) (34).

The causes of the unintentional infertility being experienced by some 2.4 million U.S. married couples are varied and difficult to pinpoint. Moreover, for some couples, infertility is a temporary phenomenon. The known and suspected causes of infertility can be grouped as environmental, pathological, heritable, iatrogenic (i.e., medication-induced), nutritional, and sociobehavioral. Birth defects afflict about 7 percent of live-born infants. As in the case of infertility, the causes of many birth defects are often unknown or speculative. Analysis of reproductive impairment in the population as a whole (see appendix A to this chapter) provides a background against which to identify any increased incidence of reproductive dysfunction that may be workplace-related.

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Chapter 4

Evidence for Workplace Hazards to Reproductive Function

CONTENTS

Page
Introduction 67

Effects of Workplace Chemicals on Reproductive Function 67
  Metals 69
  Agricultural Chemicals 74
  Polyhalogenated Biphenyls 78
  Organic Solvents 81
  Anesthetic Agents 82
  Epichlorohydrin 83
  Ethylene Dibromide (EDB) 84
  Ethylene Oxide (EtO) 85
  Formaldehyde 87
  Rubber 88
  Vinyl Halides 89
  Hormones 90
  Undefined Industrial Exposures 91

Effects of Workplace Physical Agents on Reproductive Function 93
  Ionizing Radiation 94
  Nonionizing Radiation 96
  Magnetic Fields 101
  Hyperbaric and Hypobaric Environments 102
  Hot and Cold Environments 103
  Noise and Vibration 104

Effects of Stress on Reproductive Function 105
  Psychological Stress 105
  Physiological Response to Stress 105
  Physical and Psychological Stress and the Pregnant Worker 106

Effects of Workplace Biological Agents on Reproductive Function 107
  Rubella 107
  Cytomegalovirus 108
  Hepatitis B 108
  Other Infectious Agents 109
  Recombinant DNA 109

Summary and Conclusions 110

Chapter 4 References 111

List of Chemical Names 126

List of Tables

Table No. *Page*
4-1. Estimated Ethylene Oxide Fumigation Use and Potential Operator 86
4-2. Workplace and Ambient Exposure to Formaldehyde 87
4-3. Workplace Vinyl Halide Exposures 90
4-4. Guidelines for Continuation of Various Job Tasks During Pregnancy 107

Chapter 4

Evidence for Workplace Hazards to Reproductive Function

INTRODUCTION

Two elements are required to produce a workplace reproductive hazard. First, a male or female worker, or developing embryo or fetus, must be exposed to a hazardous agent found in the work environment. Second, this exposure must compromise some aspect of male or female reproductive function, or embryonic or fetal growth and development.

This chapter reviews selected chemical, physical, and biological agents that are real or suspected workplace hazards to reproductive function.[1] These agents were chosen for review in consultation with the Advisory Panel for this report. Throughout the text, which is not a full assessment of the hazards of these agents, but rather a sumary of the evidence or lack of evidence for effects of particular agents, the focus is on available human data. These data have been integrated with animal data in order to further define the site and mechanism of action of particular adverse reproductive effects. It is important to note, however, that the identification of an agent as a suspected reproductive or developmental hazard hinges not only on its mechanism of action and evidence of harmful effects in animal and/or human data, but also on the level and kind of exposure the agent presents to humans. It is also important to point out that the National Institute for Occupational Safety and Health (NIOSH) has identified a number of occupational chemicals as reproductive hazards. These chemicals include 1,3-butadiene, carbaryl, carbon disulfide, chloroprene, dinitrotoluene, epichlorohydrin, ethylene oxide, ethylene thiourea, glycidyl ethers, glycol ethers, monohalomethanes, and polychlorinated biphenyls (PCBs).

EFFECTS OF WORKPLACE CHEMICALS ON REPRODUCTIVE FUNCTION

Of the thousands of chemicals used in the workplace, relatively few have been examined for their effects on reproductive function. A 1982 review of the reproductive hazards of industrial chemicals that explored the effects of 48 compounds (26) found significant gaps in information on reproductive toxicity in either experimental animals or humans for all but one of these chemicals. **These gaps in knowledge make estimation of human hazard difficult, and prediction of human risk virtually impossible.** Of the 48 chemicals reviewed, only a small number of those known to produce adverse reproductive effects have been classified by both endpoint and mechanism of the effect. Although reproductive toxicity has been suggested for a number of the chemicals that have been studied, many of these findings are in dispute. Moreover, some chemicals have been investigated in one sex, but not in the other. For these reasons, existing knowledge of workplace chemical hazards to reproductive function is incomplete and of uneven quality. A major conclusion of every symposium on the reproductive toxicity of suspected hazards over the past several years has been the absolute necessity for increased knowledge through additional experimental study.

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[1] A number of excellent reviews of the effects of chemical and physical hazards on reproductive function have recently been published (26,61,69,215,226,260,372,373,411).

Discrepancies among results of epidemiological studies of reproductive toxicity appear to arise from four major factors (see chapter 6):*

1. **Differences in levels of exposure of the study groups:** Exposure levels or biological indicators of exposure are frequently unknown, or not presented in research reports. In some studies, the precise identity of the chemical(s) is either not known or not revealed.
2. **Differences in accuracy and sensitivity in detecting reproductive outcomes:** The ability to detect and measure many of the endpoints of reproductive function (see chapters 5 and 6) varies from laboratory to laboratory and from country to country. For example, one laboratory may use sensitive measures of sperm motility employing video systems while another may employ the older, traditional method of watching sperm under the microscope.
3. **Definition of control groups:** The use of inappropriate controls can skew the findings of a study. For example, an investigator may compare groups composed of small numbers of participants. This can result in a finding of no adverse reproductive effects of occupational exposure. Using control groups that are not well-defined or using historical controls drawn from studies of populations with different sociodemographic characteristics may bias the results in an unpredictable direction.
4. **Confounding variables:** Failure to control for variables with the potential to modify observed effects can confound the interpretation of results. Control of these confounding variables is essential because lifestyle, ethnic, or disease-related factors may have adverse effects on male or female reproduction or fetal development (see chapter 6).

It is important to note that a majority, perhaps two-thirds, of the studies on workplace chemical hazards to reproductive function are not conducted in the United States. Most of the epidemiological studies are conducted in the Scandinavian countries and in the Soviet Union, where access to workers and workplace exposure data is less difficult than in the United States. Further, the United States has relatively few large-scale, central data bases from which both occupational and reproductive data can be retrieved. In contrast, Sweden and Finland maintain central data registries that cross-link occupational history, pregnancy data, birth certificates, medical records, and death certificates by means of an individual identification number. Until U.S. scientists have better access to occupational and health data, most conclusions regarding occupational reproductive hazards will necessarily be based in large part on studies conducted in other countries.

Reproductive toxins are classified by: 1) the site(s) or endpoint(s) of adverse effect in the reproductive system, and 2) mechanism(s) of action (see chapter 3). The site of effect defines where the compound acts to interrupt reproduction (e.g., the hypothalamus, pituitary, gonad, accessory organs, placenta, or embryo/fetus). A compound may be a reproductive toxin in the male but not in the female, or the fetus alone may be susceptible. **It is important to note that there is no biological basis for assuming that either the embryo/fetus or the female is more susceptible than the male. Only careful experimental studies and reproductive health surveillance of workers exposed to suspected compounds will provide definition of the range of human susceptibility to reproductive toxins.**

The mechanism of action of a reproductive toxin is important because it defines how the compound produces its adverse reproductive effect (226). The mechanisms of action of reproductive toxins can be classified as direct or indirect. Direct-acting reproductive toxins do not need to be processed in the body to be hazardous. A direct-acting reproductive toxin need only be delivered to its site of action to produce an adverse reproductive effect. An indirect-acting reproductive toxin, by contrast, requires some chemical change in the body before it can produce an adverse reproductive effect (see chapter 3).

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*The basic overall scarcity of data on the reproductive health effects of many of the substances summarized has led to the inclusion of research findings whose methodology or validity cannot always be determined. Gaps in information and instances of single studies for particular agents are noted where they occur.

Metals

The adverse reproductive effects of lead, mercury, cadmium, arsenic, lithium, antimony, boron, and manganese have been described in both humans and experimental animals. Other metals, such as chromium, copper, nickel, and selenium produce adverse reproductive effects in animals but have not been examined in humans. Only a fraction of the studies assessing the effects of metals on human reproductive function are framed in the context of occupational exposure to a single metal; most workplace exposures are to complex mixtures of several metals and other xenobiotics (a biologically foreign compound).

Many studies are based on workers exposed to metals while employed in metallurgical or smelting industries. These workers are often exposed to a variety of metals, as well as to other substances that may be reproductive toxins (e.g., hydrogen sulfide, sulfur dioxide). Their occupational exposure may also include such confounding exposures as heat, vibration, or dust. It is therefore difficult to attribute specific observed toxic effects in a workplace study to any single hazard, and difficult to define interactions that may increase or diminish the reproductive toxicity of any single agent.

Unlike the case of some chemical exposures, there are biological indicators of metal exposure, such as metal levels in blood, urine, and hair. In fact, the diversity of indicators often makes it difficult to reach a consensus on the toxic level for a particular indicator. For this reason, major research efforts are focused on the identification of sensitive tissues and techniques for monitoring acute and chronic exposure to metals. For some metals, such as methylmercury, there is no agreement among researchers even as to units of measurement; for others, methodology for measurement in biological samples is problematic. In hair analysis, for example, metals adhering to the outer surface of hair must be removed prior to analysis for metal content.

Metals classified by NIOSH as occupational carcinogens include arsenic, beryllium, cadmium, chromium, and inorganic and organic nickel (243). In addition, some metals (e.g., mercury, arsenic) have been found to be mutagenic to human somatic cells. This creates concern for mutagenicity to germ cells; i.e., spermatocytes and oocytes. Other metals (e.g., lead, cadmium) are capable of disrupting the cellular mechanisms involved in mitosis and meiosis, and may, by this mechanism, be toxic to germ cells.

Lead

Lead exists in the environment as a widespread contaminant in both inorganic and organic forms. Approximately 90 percent of the lead entering the atmosphere comes from the combustion of leaded gasolines. Blood levels of lead have been shown to vary directly with the content of lead allowed in gasoline (12).

Lead is found in lead azides, lead salts, tetraethyl lead, tetramethyl lead, metallic lead, tetraethylplumbane, and tetramethylplumbane. Workers who are exposed to lead include smelters, battery manufacturers, painters, typesetters, and stained glass artists. Workers may also be exposed to lead in the manufacture of paint, ink, ceramics, pottery, ammunition, textiles, and leaded gasoline.

Lead has been recognized as a reproductive hazard since the days of ancient Rome (125). Indeed, it has been suggested that lead in drinking vessels produced enough toxicity to result in the declining population of the upper class. Lead has also been used as a spermicide and as an abortifacient. Provisions for the protection of reproductive health in adults and the health of the developing embryo/fetus in the Occupational Safety and Health Administration's (OSHA) lead standard are discussed in chapter 7.

Male.—A 1975 study reported dose-related disturbances in sperm-related factors in 150 lead workers (194). A number of studies of the effect of lead on various aspects of male reproductive function were published in the 1970s (259,300, 379). One small case control study reported that 3 of 14 men had subnormal sperm counts, one patient had azoospermia, and another had low sperm motility following exposure to tetraethyl lead (379). Another study reported sexual disturbances in 66 men aged 24 to 49 who had been exposed to ethyl benzene containing tetraethyl lead. The major complaints were poor or absent erection, premature ejaculation, and reduced orgasm. Semen volume was reduced in 23 of the exposed men. A 1985 study reported no effect of lead exposure on sperm volume or motility compared with controls. Exposure ranged from 1 to 24 years in men aged 27 to 57 (347). A 1983 study of men exposed to lead found lower chromosome stability and lowered secretory function and accessory genital glands, but no difference in sperm number, motility, morphology, or semen volume (395).

There is substantial evidence of excessive rates of abnormal pregnancies among wives of lead workers. An 1860 study of 32 pregnancies in 7 women who were married to lead workers (280) recorded 11 abortions and 1 stillbirth; 8 of the 20 liveborn children died within their first 12 months. A 1985 review of similar data (374) also suggests that paternal exposure to lead alters reproductive outcome in the female. The traditional view that lead exposure leads to male reproductive problems has been supported by studies in the lead-related industries. Additional collection and analysis of data on lead exposure are needed, however, to identify other potential sites of toxicity in the reproductive system.

A 1983 review of the effect of lead on the reproductive capacity of male mammals (209) concludes that the effect of lead on reproductive function may be generally cytotoxic rather than mutagenic. The study also points out that animal data do not support the findings on human fertility. This disparity, which may reflect differences in animal/human metabolism, illustrates the difficulty in extrapolating human effects from animal studies.

Female.—Female exposure to lead has been associated with amenorrhea and other menstrual disorders, infertility, spontaneous abortion, stillbirth, and neonatal deaths (122,207,273,304,305) for more than a century and lead was at one time used to induce abortion (122). Although exposure to lead in earlier times was probably greater than it is today (46), occupational lead exposure of men and women still appears to pose a threat to normal reproductive function.

A recent review of the effects of various forms of lead on female reproduction in experimental animals noted decreased fertility, delayed vaginal opening, ovarian atrophy, and altered ovarian cyclicity (225). The sites of action include the hypothalamus, pituitary, ovaries, and uterus.

Pregnancy.—Exposure to a mixture of metals, including lead, has been associated with an increased rate of spontaneous abortion (264,265). Exposure to lead is reported to be detrimental to implantation and embryonic survival (226) and lead chloride can interfere with implantation (394). It has also been suggested that prenatal exposure to lead can result in spontaneous abortion (146). Reviews of the effects of various forms of lead on the pregnant animals (26,225, 246,394) found no teratogenic effect of tetraethyl lead, tetramethyl lead, and trimethyl lead, when given at doses below those that cause maternal toxicity.

Prenatal exposure to lead, even in small amounts, may have an effect on central nervous system development (255,302). A recent review delineates the specific pre- and post-natal periods during which particular developmental effects of lead exposure occur in the embryo/fetus (179).

Boron

Boron is used for weatherproofing wood and fireproofing fabrics. It is used in manufacturing cements, crockery, porcelain, enamels, glass, leather, carpets, hats, soaps, and artificial gems. It is also used in the manufacture of cosmetics, in printing and dyeing processes, in painting and photography, and for impregnating electric condensers and hardening steel. Boron, in the form of boric acid and borates, is widespread in the environment. Although boron is usually considered a chronic poison, effects are unlikely to be seen at an intake of less than 100 mg of boron per day.

Male.—Soviet studies (which do not describe methodology, selection of control groups, etc.) report oligospermia and decreased libido in men working in factories that produced boric acid (206,348) and in men living in communities with high boron concentrations in well water (190,206). No studies of males are available from the United States.

The major adverse reproductive effect of boron appears to be on the testes, as evidenced from studies in the rat and dog (26). Sodium borate and boric acid given orally (117, 350, or 1,170 ppm in the diet) to rats for up to 2 years caused testicular atrophy and sterility in the high-dosage group. No testicular effects were seen at 117 or 350 ppm. In a similar 2-year study of dogs fed 58, 117, or 350 ppm of boric acid, no changes were seen in histology, or in relative or absolute organ weights. High doses of 1,170 for 38 weeks caused testicular degeneration, spermatogenic arrest, and atrophy of the lining of the seminiferous tubules in the testes. Two of the dogs were put on a control diet for 25 days, after which testicular weights and spermatogenesis were found to be similar to controls, suggesting possible reversibility of the effects (387).

Female.—A three-generation reproduction study was conducted in male and female rats fed diets containing 117, 350, and 1,170 ppm boron equivalents of sodium borate and boric acid. At the highest dose level both male and female rats were sterile; the males had reduced sperm counts, and there was decreased ovulation in females. Reproduction was not affected at the two lower concentrations of boron in the diet (387).

Pregnancy.—The only studies of developmental effects available for boron involved the effects of boric acid on chick embryos (36). Injection of boric acid into chicken eggs causes growth inhibition, interference with feather growth, and several types of malformations. The relevance of these results to humans is not established, and there appear to be no published data on the effect of boron on human pregnancy. There is thus a marked lack of evidence about its reproductive and developmental effects, especially in humans.

Manganese

Manganese is present in more than 20 different compounds, including complexes with acetate, bromide, chloride, phosphate, and sulphate. It is used in the manufacture of steel, dry-cell batteries, glass, ink, ceramics, paints, rubber, and wood preservatives.

Male.—Chronic manganese poisoning in male miners has been reported to produce impotence, decreased libido, delayed ejaculation, and reduced androgen secretion (26,231,282,317). A 1985 study of 85 male workers from a factory producing manganese salts revealed markedly fewer children born to exposed workers than to nonexposed workers (202).

At doses that had no other toxic effects, there are reports of retarded growth of testes and seminal vesicles (131). The testes and accessory glands in experimental animals appear to be particularly sensitive to manganese (26).

Female.—Although one study reports depressed fertility in female rats exposed in utero (200), a recent review found no evidence of detrimental effects on females of exposure to manganese (26).

Pregnancy.—Manganese *deficiency* appears to cause developmental effects in a number of species, but there has been little study of the effects of an *excess* of manganese. Manganese appears to be harmful to the embryo/fetus only at doses that are near or above those toxic to the dam (mouse, rat, hamster, and rabbit). Postnatal development of the rodent, however, may be adversely affected if manganese is transferred from the mother to the newborns during suckling (26,216). Accumulation of manganese in the brain of the newborns may account for biochemical disturbances in the brain, as well as poor weight gain and postnatal survival.

Mercury

Mercury exists in metallic, inorganic, and organic forms, including inorganic mercury salts and organic mercury, both of which may be produced by natural processes. Humans are most likely to be exposed to these two forms of mercury from environmental contamination. The vapor of metallic mercury is the predominant form in occupational exposures. It is estimated that 40,000 U.S. workers are exposed to this form of mercury in manufacturing (e.g., electrical apparatus, mercury vapor lamps, paint, thermometers) and mining (68). Inorganic mercury appears capable of producing reproductive toxicity following ingestion, inhalation, or absorption through the skin, although the inorganic forms are less well absorbed.

The methylmercury contained in fish and fish products accounts for the balance of human exposure (68). The best documented exposures to methylmercury have not been in the workplace, but in the home, through the ingestion of contaminated fish (see chapter 2) or seed grain.

Male.—Both organic and inorganic mercury can alter spermatogenesis and decrease fertility in experimental animals (26). Altered libido has been observed in men accidentally exposed to mercury vapor. In experimental animals, organic mercury also accumulates in the central nervous system in regions that are involved in the control of reproduction. This suggests that occupational exposures to metallic, inorganic, or organic mercury may disrupt male reproduction at multiple sites.

Female.—Various forms of mercury accumulate in the ovary of experimental animals; inorganic mercury preferentially accumulates in the granulosa cells surrounding oocytes, while metallic mercury accumulates in the corpus luteum (225). Accumulation of mercury in the central nervous system is consistent with the menstrual disturbances observed in women following occupational exposure. Monkeys treated with mercury also show alterations in hypothalamic, pituitary, and ovarian function.

Pregnancy.—Inorganic and organic mercury can cross the placenta and gain access to the fetus in both animals and humans. In experimental animals, metallic mercury and inorganic mercury alter fetal growth, increase fetal mortality, and increase the incidence of congenital malformations. Mercury can also produce biochemical changes in the human placenta. Mercury, used historically in the treatment of syphilis, has also been associated with an increase in spontaneous abortions among women treated during pregnancy. The data on organic mercury also show evidence of developmental effects in both humans and experimental animals (69).

All forms of mercury appear to be reproductive toxins. Sites in the reproductive system that are impaired include the hypothalamus, pituitary, and gonad. Effects include chromosome abnormalities, increased rates of spontaneous abortion, low birth weight, congenital malformation, and abnormal development of the nervous system.

Cadmium

Cadmium is used in industry for corrosion protection, as a plastics stabilizer, for electroplating, and in nickel-cadmium batteries, pigments and paints, soldering liquids, semiconductors, photocells, insecticides, and fungicides. Cadmium is set free during welding. Although under some circumstances occupational exposure is the dominant source of exposure, the major source of cadmium intake is usually food (113). Cadmium occurs naturally in zinc-bearing minerals and in phosphate rocks, which are used to make many fertilizers. Cadmium absorption thus occurs from food, water, and air (339). One pack of cigarettes contains 30 micrograms (μg) of cadmium (78), and smoking may contribute to half of the total body cadmium when occupational exposure and exposure via food are low (113).

Some studies have indicated an increased frequency of chromosomal aberrations following exposure to cadmium while others have not (50, 276,327). The chromosomal damage observed in several studies may be attributable to lead exposure, cadmium exposure, or the synergistic effects of exposure to both metals (31,79). Cadmium is classified as an occupational carcinogen, and may therefore alter the integrity of germ cell DNA in workers.

Male.—The testicular toxicity of cadmium has been conclusively demonstrated in experimental animals (26,188,279,292). The effect appears to result from the direct toxicity of cadmium to testicular capillary lining. Human exposure to cadmium fumes or dust is also associated with testicular toxicity, altered libido, and infertility (26).

Female.—Although cadmium has been demonstrated to accumulate in the ovary of experimental animals, there are no reports of alterations in human female pre-implantation reproduction. Women exposed occupationally to cadmium appear to have normal integrated function of the hypothalamus-pituitary-ovarian axis (411).

Pregnancy.—Cadmium impairs implantation and produces placental necrosis in experimental animals. Similar effects on placental vasculature have been reported in women exposed to cadmium. In addition, occupational and environmental exposure to cadmium have been associated with decreased birth weight (69,411). Congenital malformations have been observed in experimental animals following cadmium exposure. However, it is not known whether human exposure is associated with a higher frequency of congenital malformations.

Arsenic

Arsenic occurs in industry largely as a byproduct of copper and lead smelting. It occurs naturally in trace amounts in soil, minerals, and some foods. Compounds containing arsenic are used in pesticides, glass, ceramics, paints, dyes, wood preservatives, and leather processing. An estimated 545,000 workers in the United States are potentially exposed to arsenic in metal smelting and in the manufacture and application of pesticides (112).

Male.—Evidence of an adverse effect of arsenic on male reproductive function is inconclusive (316). Workers exposed to arsenic at a smelter in northern Sweden were found to have an increased frequency of chromosomal aberrations when compared with healthy males from a nearby city. Among the affected smelter workers, the groups with higher exposure to arsenic had a greater frequency of chromosomal aberrations. The data also suggested an interaction between smoking and arsenic exposure, although smoking status was not controlled in the analysis (263). An increased frequency of chromosomal aberrations was found in the white blood cells of wine growers exposed to arsenic pesticides (263) and in patients with psoriasis treated with arsenic (53).

Recent studies list several effects of arsenic on reproductive function in mice and pigs, including testicular toxicity, altered sexual behavior, and impaired sperm quality and fertility (26). Effects are seen only at higher levels and the decreases in fertility are probably secondary to abnormal sexual behavior.

Female.—Studies of the effect of arsenic on the female have largely been limited to its carcinogenic potential. No effects on the fertility of female mice in multigeneration studies at doses ranging from 0.025 to 215 mg/kg of diet have been observed (26). Although arsenic has an effect on post-fertilization events, it apparently has no direct effect on the mature reproductive system (226).

Pregnancy.—A 1982 study examined the rate of spontaneous abortion in a Scandinavian community where a metallurgic industry was located (144). The industry produced mostly zinc and cobalt and emitted sulfur dioxide, hydrogen sulfide, arsenic, and to a lesser extent, cadmium and mercury into the environment. Twenty-five percent of the community's men were employed at the metals plant. The wives of workers in the metallurgic industry had a higher rate of spontaneous abortion (11.5) than wives of all industrial workers (9.3 percent). This study also demonstrated that specific male and female occupations may provide increased risk of adverse pregnancy outcome.

Several other studies of female workers in the metallurgy industry in Finland, who were exposed to arsenic as well as sulfur, zinc, cobalt, and copper, were based on women who were members of the Metal Workers Union between 1973 and 1976 (141). The rate of spontaneous abortion was found to be higher among the 35,000 metal workers (13.8 percent) than in the general population of Finnish women (10.3 percent). Parity was not factored into the data analysis. A 1983 update of this study that included membership up to 1979 (146) reported no difference in the rate of spontaneous abortion for pregnancies before or after union membership (7.1 percent). Spontaneous abortions were more frequent among smelters (21 percent) than among other union members, but the numbers of workers studied was small ($n=7$).

Inorganic arsenic in the pentavalent (arsenate) or trivalent (arsenite) form is fetotoxic and teratogenic to rodents (154,155,156,245). Of the two forms, arsenate has been the most extensively studied, and at doses equally toxic to the mother produces the highest malformation rate. Arsenite is more toxic than arsenate, however, and thus is teratogenic at lower doses. The inorganic arsenicals produce a broad spectrum of developmental toxic effects, ranging from inhibition of fetal growth and prenatal death to gross skeletal malformation, including neural tube defects such as exencephaly (brain outside of the cranial cavity). A single intraperitoneal injection of sodium arsenate at 45 mg/kg body weight of pregnant mice on day 8 of gestation resulted in a 65-percent incidence of exencephaly (245). Higher doses (60 or 75 mg/kg/body weight) produced significant maternal toxicity. Sodium arsenate and sodium arsenite are considerably less toxic and teratogenic when given orally than when given by intraperitoneal injection. In the case of arsenite, doses required to produce fetotoxicity and maternal toxicity are similar. Organoarsenicals (e.g., methylated arsenicals such as sodium cacodylate) are significantly less toxic to the rodent embryo than are inorganic arsenic compounds (155).

Antimony

Salts of the trivalent and pentavalent forms of antimony, which have been used for centuries as drugs, have more recently been used as parasiticides (30). Metallic antimony is used in some alloys and inorganic salts are used as pigments, abrasives, and flame retardants.

There is little evidence that antimony acts as a reproductive toxin in either humans or animals. Although radioactive antimony is released from nuclear industries, it does not appear to be a teratogen, probably due to its inability to cross the placental barrier. Antimony can be passed to offspring via the milk of the exposed mother (123).

A Russian study found that women working in an antimony metallurgy plant had a higher incidence of premature births, spontaneous abortion, and other, unnamed reproductive system disorders. Their infants did not gain weight as rapidly as infants of nonexposed women (34). Further experimental data will be required before antimony is judged to be toxic or nontoxic to the reproductive system.

Agricultural Chemicals

Agricultural chemicals include compounds used as insecticides, herbicides, and fungicides. Certain of these chemicals (i.e., dibromochloropropane (DBCP), Kepone (chlordecone), and 2,2-bis [p-chloro-phenyl] 1,1,1-trichloroethane (DDT) are no longer used in the United States, in part because of adverse reproductive effects in animals or humans. Nonetheless, these chemicals are important to consider because of: 1) their similarity to chemicals still in use; 2) their long-term effects on workers who were exposed to them during their production and use; 3) their possible persistence in the environment; and 4) their sites and mechanisms of action, which have undergone detailed investigation and can provide useful insights into reproductive toxicity.

Exposure to agricultural chemicals can occur throughout the manufacturing process of these products as well as during their distribution, sales, and final application. Few agricultural chemicals are well-studied. In some cases there has been only one animal or human reproductive investigation of a given chemical. In most cases only one or a small number of reproductive variables have been studied for each compound. The reproductive outcomes that have been studied are usually in males. There is a notable lack of data on the effects of exposure of women workers to agricultural chemicals in the English literature, although several studies conducted in eastern Europe and Russia suggest the potential reproductive toxicity of these substances.

Although agricultural chemicals have been shown to have a variety of reproductive effects, published studies do not provide good evidence of individual human exposure levels to a given chemical. Several studies have utilized aggregate, rather than individual, data. Although this approach is appropriate for early studies designed to identify reproductive hazards, it may not be useful for deriving definitive conclusions about effect or causality. Unfortunately, individual exposure levels are difficult to secure in the agricultural chemical field because of a lack of industrial hygiene data and inadequate long-term exposure records. It is even more difficult to gauge exposure in circumstances where exposure

Photo credit: Pemina Meisels

Further study is needed of the unknown reproductive and developmental chemicals that are similar to DDT and DBCP, which have been banned in the United States.

occurs outside the production site; for example, to the pesticide applicator. Despite these difficulties, evaluation of animal and human data implicates selected agricultural chemicals as reproductive toxins and suggests the need for further animal studies of the reproductive effects of these economically important compounds.

Carbaryl

Workers may be exposed to carbaryl (1-Napthyl methyl carbamate), a broad-spectrum insecticide, during both its manufacture and its widespread application. It is readily absorbed through the skin. The potential for exposure during the manufacturing process is probably greatest among workers bagging the product (404).

Male.—Animal studies have demonstrated that carbaryl is distributed to the testis, seminal vesicles, and prostate after absorption. Suggestive data link carbaryl exposure and male infertility, although a definitive relationship has not yet been established. Chronic feeding of carbaryl to experimental animals impairs spermatogenesis and fertility and produces testicular atrophy. In 1979, carbaryl-exposed workers were compared with nonexposed workers with respect to sperm count and blood levels of reproductive hormones. No abnormalities in blood or semen could be related to carbaryl. A borderline decrease in sperm count was observed among carbaryl-exposed workers (393). A reexamination of the same cohort of carbaryl-exposed workers 2 years later identified an excess of morphologically abnormal sperm compared with the sperm of nonexposed, newly hired employees (404).

Female.—There has been little study of the effect of carbaryl on the female reproductive system in humans or experimental animals. Other cholinesterase inhibitors[2] have been demonstrated to alter reproductive function in experimental animals and are associated with reproductive abnormalities in exposed populations. Women exposed to cholinesterase inhibitors in agricultural chemical production or application have an increased incidence of menstrual cycle disturbances and secondary infertility. Data from acute poisoning suggest a direct effect on the ovary.

Pregnancy.—Carbaryl has been demonstrated to be a structural teratogen in experimental animals. However, the doses required are close to those that are lethal to the maternal organism. Its effects on the human embryo/fetus are unknown.

Dibromochloropropane

Dibromochloropropane (DBCP), a nematocide, was widely used in agriculture in the United States and abroad from the mid-1950s until 1977. In 1977, the discovery of adverse reproductive effects in humans led to a partial ban on its production in the United States. Prior to the ban, DBCP was used on a variety of crops, including cotton, soybeans, fruits, nuts, vegetables, and ornamental plants. Since 1981, the sole U.S. use of DBCP has been on Hawaiian pineapple plantations. The pineapple industry won a reprieve after promising to reduce worker exposure to the chemical. In 1985, the Environmental Protection

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[2] Choline esters transmit information between nerve cells. Cholinesterase metabolizees choline esters to maintain proper levels of the choline esters in the body. Cholinesterase inhibitors prevent the metabolism of choline esters and thus permit abnormal levels of the esters to accumulate in the body.

Agency (EPA) mandated that remaining uses of DBCP in Hawaii be phased out by 1987. DBCP has been found in drinking-water wells on Oahu and Maui (380,383). (See chapters 2 and 7 for further detail on DBCP.)

Male.—Interest in the adverse human reproductive effects of DBCP arose in the late 1970s when DBCP production workers in a northern California chemical plant complained of their inability to father children. Initial studies (391) confirmed semen and hormonal abnormalities in 11 of the 25 men who had not had vasectomies, and found a direct relationship between sperm count and duration of DBCP exposure in the others. When divided into groups by duration of exposure, 9 of 11 men with the longest exposure (an average 8 years) were azoospermic and two had sharply reduced sperm counts with reduced motility and increase in abnormal forms. Subsequent studies of 154 DBCP-exposed and 42 nonexposed workers in this plant confirmed the original findings of testicular toxicity (235,316,392).

Animal studies confirm the specific toxic effect on the testes. In the 1960s, prior to the observations of the effects on male pesticide-manufacturing workers, a comprehensive, multispecies study demonstrated the testicular toxicity of DBCP (353). In this study, testicular atrophy in rats was noted even at the lowest of three dose levels. Later studies confirmed these effects in rats and rabbits (52,296,297).

Eventual recovery of spermatogenesis following DBCP-induced testicular toxicity has been documented in some but not all of the exposed men. In Israel, 4 years after DBCP exposure, 17 healthy children were born. However, the sex ratio in this group was highly abnormal. Only 6 of the 17 (35 percent) were males (the ratio is normally 105 males for every 100 females). A subgroup of men who had recovered from azoospermia and oligospermia showed an even more skewed sex ratio of 2 males in 12 live births (16.6 percent).

Female.—DBCP has been shown to alter ovarian function and decrease fertility in female animals (297). Although females have been less thoroughly studied than males, females appear to be less sensitive to the toxicity of DBCP. Its effect on human female reproductive function is not known.

Pregnancy.—There is some evidence of fetal weight reduction in rats (310).

DBCP is clearly a testicular toxin in men and experimental animals. The extent of damage is proportional to the extent of exposure. The effects of DBCP on female reproduction and pregnancy in animals and humans require further investigation.

DDT

DDT (2,2-bis(p-chloro-phenyl)1,1,1-trichloroethane) is a pesticide in common use around the world. It reached its peak agricultural use in the United States in 1959, but U.S. use was halted in 1972 in response to concern about the pesticide's wide-ranging effects on the ecosystem. Because DDT accumulates in fatty tissue, its presence persists in the body for many years. Major concern about the reproductive toxicity of DDT arose because it mimics the effects of estrogen, a normal sex steroid in males and females.

Most of the animal studies that have been conducted on the effects of DDT have been multigeneration reproduction studies on the rat, mouse, rabbit, and dog. Chronic exposure to DDT impaired fertility in female rats and caused reduced weight gain and survival of the offspring. In the dog, administration of DDT caused early onset of estrous but all other fertility parameters were normal. With a 14-month regimen, male dogs experienced diminished libido and females had delayed estrous, infertility, and increased infant and maternal mortality.

Rabbits exposed to DDT exhibit premature delivery, increased fetal resorptions, and decreased intrauterine growth but show no evidence of teratogenic effects (260).

The effects of DDT on avian eggshells (DDT decreases eggshell thickness) are a direct reflection of its estrogenic properties. DDT can also increase the metabolism and excretion of estrogen. This is thought to partially explain the lack of calcium metabolism and soft egg shells in birds of prey (281).

In a comprehensive study of the health effects of DDT exposure of migrant farm workers, menstrual irregularities were the most frequent complaint of women seen in health clinics (63).

In addition to its adverse effects on the adult reproductive system, DDT exposure alters the development of the reproductive system. Human prenatal exposure to DDT has been suggested to be associated with polycystic ovary disease. Other systems of the developing organism may also be susceptible to adverse effects following prenatal exposure to this estrogen.

DDT has been found as a contaminant in human breast milk in persons exposed both occupationally and otherwise. However, no association has yet been found between milk concentrations and human health effects of DDT (397).

Mutagenic properties of DDT were studied in Brazil in 23 DDT-production workers and 35 nonexposed persons. Exposure levels were quantified by measurement of plasma levels of DDT and its metabolic products. This study showed a higher frequency of white blood cells with chromosomal abnormalities among workers with high blood DDT levels than among those with low blood DDT levels (294).

Kepone [Chlordecone]

Kepone is a chlorinated hydrocarbon insecticide and fungicide that mimics the action of estrogen and is chemically related to Mirex, Endrin, Dieldrin, Heptachlor, chlorophenothane, and DDT. Kepone was manufactured and used in the United States until 1975. Its use was banned in 1977. Kepone was used most commonly as a pesticide against fire ants and in ant and cockroach traps.

Male.—Reported effects of Kepone on male fertility include reduced sperm count and motility and decreased spermatogenesis as judged by testicular biopsy in 13 of 23 exposed Kepone production workers (349). Abnormal sperm morphology has also been reported in Kepone production workers (56). Animals exposed to Kepone exhibit adverse effects on the testes at doses as low as 10 ppm in the diet over a prolonged period (2 years) (96).

Female.—Female rats and mice fed Kepone in the diet exhibit constant estrus with some damage to the ovaries (134,157). No human studies are available.

Pregnancy.—Kepone has been shown to alter embryonic development in animals but at levels that are also toxic to the dam (67). Female offspring that survive prenatal or neonatal treatment suffer reduced reproductive capacity (102,120, 121). There is evidence that Kepone can concentrate in breast milk in humans (124,159). No data on developmental effects in humans are available.

2,4,5-T, Dioxin, and Agent Orange

2,4,5-T (2,4,5-trichlorophenoxyacetic acid) is a chlorinated herbicide that was used widely in the United States from 1948 until 1970 in large-scale farming, family gardens, forest management, and weed control along roadsides and railroad rights-of-way. The observation of birth defects in animals exposed to 2,4,5-T led the U.S. Department of Agriculture to suspend many uses in 1970. In 1979, EPA banned the use of 2,4,5-T except for range land and rice fields.

In 1957, **dioxin** was identified as a contaminant of the synthesis leading to 2,4,5-T. Dioxin (2,3,7,8-tetrachlorodibenzo-*p*-dioxin, or TCDD) also occurs as a contaminant in the manufacture of 2,4,5-trichlorophenol (TCP), which, in turn, is used in the synthesis of 2,4,5-T and 2-(2,4,5-trichlorophenoxy) proprionic acid, also known as Silvex. Dioxin, then, is an unwanted, unavoidable contaminant in the manufacture of these other chemicals. It is not a product in itself.

NIOSH reported in 1984 that it was not possible to provide an accurate estimate of the number of U.S. workers then at risk of exposure to dioxin (370). Occupational exposure to dioxin may occur:

- during production of TCP;
- in decontamination of worksites from prior production or use of TCP, 2,4,5-T, or Silvex;
- from waste materials, such as reclaimed oil, contaminated with dioxin;
- from cleanup after fires in transformers containing polychlorinated aromatics; or
- from dioxin-contaminated dust or soil particles that can remain airborne or accumulate on indoor or outdoor work surfaces.

Agent Orange was the most widely used of several herbicides sprayed by U.S. military forces for defoliation and crop destruction between 1962 and 1971 during the Vietnam War. Most of the spraying was done between 1967 and 1969 from fixed-wing aircraft, as part of "Operation Ranch Hand." Agent Orange was a 50/50 mixture of 2,4-D (to be discussed) and 2,4,5-T (114).

Public concern over possible reproductive effects of Agent Orange has been extreme for three reasons. First, between 2.4 and 2.8 million American military personnel served in Vietnam, and an unknown large number of Vietnamese soldiers and civilians lived or fought in sprayed areas. Second, anecdotal reports persist of birth defects attributed to exposure to Agent Orange or its constituents. Third, Agent Orange contains 2,4,5-T, which is contaminated during manufacture by dioxin (114).

Males.—Definitive adverse reproductive effects of occupational exposure to 2,4,5-T or dioxin on adult reproductive function have not been documented. To date, studies of exposed and nonexposed groups of workers have found no differences in semen characteristics, male potency and libido, infertility, and spontaneous abortion (201, 331,342).

A study of U.S. Air Force personnel who worked with Agent Orange in Vietnam found an excess of minor birth defects, such as birthmarks, among their offspring compared with the offspring of nonexposed personnel. No difference in incidence of more severe birth defects was observed between the exposed and nonexposed groups. In this study, the Air Force Ranch Hand Study, data were obtained from parental history and were not verified through medical records (130,201). A study based on the experiences of parents of babies born in metropolitan Atlanta from 1968 to 1980 contained no evidence to indicate that Vietnam veterans have been at greater risk than other men for fathering babies with birth defects, when all types of serious structural birth defects are combined (97).

Although concern about the effects of dioxin on the offspring of exposed males has overshadowed concern about the direct reproductive toxicology of dioxin, there is little or no evidence to suggest that dioxin alters fertility or sexual function in human males (355).

Female.—Female reproduction in animals appears to be sensitive to dioxin. At doses of 1 μg/kg/day for 13 weeks there were changes in estrous cyclicity and corpora lutea formation (184). There is also evidence of altered steroid metabolism and/or production in nonhuman primates exposed to dioxin (27).

2,4-D

2,4-D (2,4-dichlorophenoxyacetic acid) is an herbicide commonly used in agriculture and forestry. It is closely related to 2,4,5-T in chemical structure. A 1984 case report from Arkansas described multiple malformations, including facial, digital, and limb defects and severe mental retardation, in a child born to parents who had both been heavily exposed to 2,4-D while spraying trees (59). Exposure of the parents was prolonged and at high levels and occurred both through respiratory and cutaneous routes. Exposure to the mother occurred 7 hours per day, 6 days per week from 6 months before conception to 5 weeks after her last menstrual period, when pregnancy was confirmed. A study of rats exposed prenatally on days 6 to 15 of gestation, reported subcutaneous edema, wavy ribs, delayed ossification, and lumbar ribs (319).

Polyhalogenated Biphenyls

Polybrominated biphenyls (PBB) and polychlorinated biphenyls (PCB) belong to a class of chemicals known as halogenated aromatic hydrocarbons. They have been a valuable resource in industry because of their chemical stability, low volatility, and nonflammability (210). Yet these same properties cause the persistence of these chemicals in the environment. They are a potential reproductive health concern to humans and animals because once absorbed they are metabolized poorly, excreted slowly, and accumulate in fatty tissue (309). Since 1979, all manufacture, processing, and distribution of these chemicals has been banned in the United States, in part out of concern for reproductive toxicity (244).

There is a dearth of information concerning the reproductive effects of PBB and PCB, and existing information is derived largely from incidences of food contamination rather than workplace exposure. Both PBB and PCB are known to cross the placenta, although not readily. Levels of these agents, which are extremely fat-soluble, have been found in breast milk at up to 100 times maternal blood levels. Lower birth weights and dermatological effects of PCB and PCB-like chemicals were observed in offspring of women exposed in a cooking-oil contamination accident in Japan, but no persistent morphological or behavioral effects have been documented. There have been no reports of congenital malformations associated with PBB.

Polybrominated Biphenyls

Polybrominated biphenyls (PBB) were developed for use as a flame retardant in thermoplastic products (309). In 1973, PBB was inadvertently mixed into cattle feed in Michigan, which led to widespread contamination of the food chain (176). Measurement of PBB in breast milk suggested that the chemical had been widely disseminated across the State (47), a finding later confirmed in a larger study (398). Farmworkers exhibited higher PBB levels than the general population (397), but there were few objective findings related to male reproductive effects (316). Most of the information on the health effects of PBB has been generated from this Michigan incident.

Male.—To date, only one human study has examined the effects of PBB on human spermatogenesis. Research efforts generated in 1979 by findings of a clinical field study of PBB-exposed men who complained of loss of libido (11) analyzed the semen quality of farmers and individuals who had consumed food from PBB-contaminated farms and PBB workers who might have inhaled or ingested the chemical (309). The results showed no difference in sperm counts, motility, and morphology in these men compared with a control group of male university students. Men with possible confounding factors (e.g., varicocele; marijuana use) were eliminated. However, because the collection of sperm did not occur until 4 years after the contamination incident, an earlier transient effect of PBB on spermatogenesis could have been reversed. No other studies have been conducted to explore the effect of PBBs on human male reproduction (26).

Although testicular damage and abnormalities in sperm function have been reported in cows and monkeys, these effects appear to be secondary to the general toxicity of this compound (26). Polybrominated biphenyls are also potential inducers of the hepatic mixed function oxidase system, which might alter testosterone pharmacokinetics and indirectly impair testicular function.

Female.—Disrupted menstrual cyclicity and a 7 percent weight loss were observed in monkeys fed 0.3 ppm PBB; no other signs of toxicity were observed (4). Perinatal exposure to PBB increased liver metabolism of estrogens in offspring of rats. The effect of estrogen on uterine weight and uterine RNA content was also decreased (41).

Pregnancy.—A 1983 analysis of blood, placenta, and umbilical-cord blood samples, as well as tissue and milk samples, from women giving birth found that cord blood and the placenta contained one-tenth the maternal serum concentration of PBB (103). In a 1984 study (165), cord blood contained one-sixth the maternal serum concentration of PBB.

The high fat solubility of PBB allows it to accumulate in maternal breast milk. Detectable levels of PBB were found in 96 percent of the 53 samples randomly collected from nursing mothers in Michigan's lower peninsula (47). In another study, breast milk levels of PBB in women living on PBB-contaminated farms were more than 100 times greater than their blood levels, and reached approximately 80 percent of the PBB level in their body fat tissue (103). In a 1984 study, breast milk levels of PBB were twice those of maternal blood (165).

A number of studies have been conducted to assess the possible effect of PBB exposure on the developmental abilities of young children (318, 320,386). The studies examined children in Michigan who were exposed to PBB in utero, in early infancy, or both. A number of these children were breastfed by mothers who ingested PBB-contaminated foods. The first investigation of this kind, in 1981, failed to identify any effects of PBB on physical health and growth when 33 children born on PBB-contaminated farms were compared with 20 unexposed controls. Psychological development tests were also negative. However, on several of the McCarthy Scales of Children's Abilities an inverse relationship was shown between body-fat PBB level and performance. The mean age of the children was 37.2 months (386).

Developing fetal and newborn animals are readily exposed to PBB by transplacental and milk transfer from the exposed mother (25,88). Placental transfer of PBB has been shown in the cow, rat, and guinea pig.

PBB administration to pregnant rats causes lower body weight, increased mortality, and liver carcinomas in the offspring (132). Feeding PBB to pregnant pigs causes toxicosis in the dams and abnormalities in the thyroid and liver of the offspring. The major route of exposure of the offspring appears to be via the mother's milk. PBB can also cross the placenta in the pig (388).

Polychlorinated Biphenyls

Polychlorinated biphenyls (PCB) are a family of synthetic compounds introduced in industry in 1929. Until the 1970s, these chemicals were manufactured and used in coolant fluid in electrical transformers, hydraulic fluids, lubricants, plasticizers, coatings, sealants, and pesticide extenders. Mixtures of PCB may be oily, viscous liquids, or sticky resins.

PCB may enter the workplace or ambient environment through the careless disposal of industrial fluids, the leakage of nonclosed systems, and electric transformer fires. PCB has been found in samples of air, soil, water, and fish. Since the 1979 EPA ban on manufacturing, processing, and distribution of PCB, occupational and environmental exposure has been reduced (210). The principal hazard today rests with transformers and capacitors put in use before the ban and still containing PCB fluid. Estimates of the number of PCB-containing transformers range from 20,000 to 150,000 (65). PCB-laden transformers pose a potential hazard to utility workers, appliance service workers, and fire fighters (210).

Males.—There are no reports of studies designed to evaluate the effect of PCB on human male reproduction (26,316). Postnatal exposure to PCBs depresses mating ability and fertility in adult male rats (311). Male reproductive function appears to be somewhat resistant to the effects of PCB (26).

Females.—Women exposed to high levels of PCB have been reported to experience altered menstrual cycles (384). Chronic exposure to 5 ppm in female mice and monkeys causes prolongation of the estrous cycle. Ovulatory failure has also been observed in exposed female monkeys (26,28). Daily exposure of rats to 30 mg/kg Aroclor 1254 for 1 month produced prolongation of the estrous cycle, decreased sexual receptivity, vaginal bleeding during pregnancy, decreased litter size, and delay in the time to parturition (45).

After 18 months of consuming 2.5 to 5.0 ppm PCB, female rhesus monkeys were placed on a control diet for 1 year. Infants born to these mothers showed signs of PCB toxicity similar to those of siblings born during PCB intoxication. This illustrates the tremendous residual ability of PCB in the female (2). The reproductive effects of PCBs in mammals include longer estrous cycles, decreased implantation sites, and increased stillbirths in a variety of species, including rats, mice, rabbits, monkeys, dogs, and mink (178).

Pregnancy.—Several studies indicate pregnancy abnormalities in women exposed to high levels of PCBs following the ingestion of contaminated rice oil (26). A recent study reports that pregnant women with Yusho (rice oil disease) deliver babies with fetal PCB syndrome (407). The symptoms include dark brown pigmentation, gingivial hyperplasia, shorter gestation length, and lower birth weight. The study's authors suggest a possible alteration in calcium metabolism similar to that seen in the fragile egg-shell formation exhibited by DDT-exposed birds.

Women exposed to PCB 3 to 4 years prior to conception have high levels of placental monoxygenases, enzymes that are capable of metabolizing many environmental pollutants to reactive products that may be toxic to the fetus (400). These findings suggest that PCB stored in maternal adipose tissue could have a persistent effect on placental metabolism in subsequent pregnancies. An inverse correlation between PCB exposure and fetal head circumference and birth weight has also been reported (108).

A number of abnormalities of pregnancy have been associated with PCBs in animals (26). The effects include disruption in implantation and prolonged gestation. PCB does not appear to be teratogenic or fetotoxic when given after implantation. Behavioral effects have been noted in mice exposed prenatally to PCBs. Neonatal exposure to PCB through the milk has been shown to impair the fertility of male and female offspring (26). An interesting interaction between dioxin and PCB has been reported in which PCB potentiates the dioxin-dependent cleft palate formation in mice tenfold (37). This suggests that exposure to complex mixtures in the occupational environment may be more harmful than exposure to individual compounds.

Both PCBs and PBBs appear to be reproductive toxins in both male and female; fetal toxicity may also occur. Because PCBs and PBBs are metabolized very slowly, exposure may exert adverse effects even when it is far removed in time from reproduction.

Organic Solvents

Organic solvents such as carbon disulfide, carbon tetrachloride, styrene, xylene, toluene, and benzene are widely used in manufacturing and in the chemical industry. A new, major source of potential occupational solvent exposure is the electronics industry, where these chemicals are used to clean and fabricate electronic components. Despite the potential daily exposure of an estimated 10 million workers to organic solvents, few studies have examined the reproductive effects of these chemicals. Many solvents are mutagenic and carcinogenic in experimental animals, and some have been identified as human carcinogens. Carbon disulfide has been identified as an occupational reproductive hazard by NIOSH (244).

Accurate biological indicators of most solvent exposures, such as urine or blood levels, unlike those for some metal or pesticide exposures, can only be obtained soon after exposure because of the rapid metabolism and clearance of the chemicals. Many of the workers studied were exposed to multiple solvents and often to other chemicals. Little is known about the synergistic effects of multiple exposures that include industrial alcohols.

Studies on the neurotoxicology of solvents suggest the existence of a synergistic relationship between alcohol use and solvent exposure, yet no studies on the reproductive hazards of solvents have factored alcohol use into the results. Nor have other confounding variables been taken into account in analysis of the data. Most of the reported results are therefore based on crude estimates of actual exposure.

Male.—It is likely that solvents affect male fertility and semen quality. Single studies of carbon disulfide and derivatives of toluene have reported deleterious changes in semen quality, levels of serum FSH and LH, and testicular size (1,133,316). Wives of workers exposed to carbon disulfide have an increased rate of spontaneous abortion (141), and wives of painters exposed to aromatic solvents were found to be more likely to have children with congenital malformations. The effects of benzene, carbon tetrachloride, styrene, trichlorethylene, and xylene on male fertility in humans have not been investigated.

Some information on male reproductive effects of solvents is available from animal studies. Carbon tetrachloride produces testicular atrophy in mice and rats (172,321) Trichlorethylene has recently been examined for male reproductive effects in animals (410). No structural changes were observed, but reproductive behavior was altered. Male rodents may be more suspectible to exposure to carbon tetrachloride than females (26). There have been no studies of the effect of benzene on male fertility except for one dominant lethal study (26). Carbon tetrachloride is carcinogenic in several animal species, increasing concern for germ cell mutations. No effects on fertility and no dominant lethal effects were observed in one study of the effect of styrene on male mice. The effects of xylene have not been studied.

Female.—Adverse reproductive effects have also been observed in women workers exposed to organic solvents. Irregular menstrual flow has been associated with carbon disulfide exposure (55,93). A recent study of women workers found no association between styrene exposure and menstrual disturbances, refuting the findings of an earlier study (208). An increase in the incidence of spontaneous abortion has been associated with carbon disulfide exposure (144), and inconsistently associated with styrene exposure (141). Three studies have reported increased incidence of toxemia in solvent-exposed women (carbon disulfide, styrene, and mixed solvents) (55). Menstrual disturbances and heavy bleeding have been observed in women exposed to benzene, and women appear to be more susceptible to benzene exposure than men (160).

A 1975 report noted adverse effects on the estrous cycle of female rats (16) exposed to benzene; confirmation is needed from other studies. Effects of carbon tetrachloride on estrous cycles in rodents have been inconclusive because the relationship of the general toxic effect on liver function to gonadal function is unclear. No work has been done to ascertain whether there are similar effects on males (26). Inhalation exposure of the rat to styrene appears to alter gonadotrophic function and estrous cycles; the levels of exposure, however, are just below those which cause overt toxicity (26,163,412). No data are available for toluene and xylene.

Pregnancy.—Several studies have suggested that children of solvent-exposed workers are more likely to have congenital malformations and tumors; three studies have implicated solvent exposure in malformations of the nervous system. One study suggests the existence of a fetal solvent syndrome similar in nature to the fetal alcohol syndrome; because the structure and metabolism of many industrial alcohols are similar to those of ethanol, such a solvent syndrome is considered plausible (151,152,192,274,354). Studies are needed on exposure during pregnancy to confirm or deny this effect. Benzene crosses the placenta and is present in fetal blood in amounts equal to or greater than levels in maternal blood (84). No data are available for carbon tetrachloride.

Benzene and carbon tetrachloride may alter ovarian function in experimental animals (16,26). Consistent findings on benzene's effects during pregnancy in the mouse, rat, and rabbit include embryolethal and teratogenic effects such as reduced body weight and skeletal variants in the offspring at doses that are not toxic to the dams (26,158,247,385). The industrial solvent 2-ethoxyethanol is a behavioral teratogen in rodents; human effects have not been defined (356).

Anesthetic Agents

At room temperature, anesthetic agents are either gases or volatile liquids. Traces of anesthetics present a potential occupational health hazard when these gases and vapors leak from the anesthetic breathing circuit. An estimated 214,000 medical personnel, including surgeons, anesthesiologists, nurse anesthetists, operating room nurses and technicians, dentists, laboratory personnel, and veterinarians are regularly exposed to anesthetic agents (362).

The most widely used anesthetic gas is nitrous oxide (375). Other commonly used agents include fluorinated hydrocarbons (halothane, enflurane, and methoxyflurane) and cyclopropane. The fluorinated hydrocarbons replaced diethyl ether and chloroform, which were used commonly as anesthetics until 1950 (362). While dentists tend to administer nitrous oxide alone, physicians primarily use nitrous oxide in combination with the halogenated agents, making the effect of any one agent difficult to document (73). Levels of waste anesthetics in ambient air depend on: 1) anesthetic technique, 2) scavenging devices, and 3) ventilation systems (375).

There is concern for two undesirable reproductive outcomes in humans with occupational exposure to anesthetic agents: 1) an increase in the frequency of spontaneous abortion, and 2) an increase in congenital malformations (147,162,316). The various epidemiologic investigations are difficult to compare and to validate because they lack information on the actual chemical agents used and quantification of exposure. Most of the studies define "exposure" by occupation—for example, operating-room nurse, dentist, or anesthesiologist—and/or by number of years spent working with anesthetic agents. Further, few studies have discussed the sorts of scavenging devices or ventilation systems, or lack thereof, operating within the workplace.

General methodological problems characterize many of the studies (89,109,147,162,377,378). Pitfalls include retrospective design and the use of poorly designed postal questionnaires, the primary source of data for most studies. A common criticism is the degree of candor of the questionnaires: they were often considered to be "loaded" so as to encourage a bias in reporting. For example, one study (9) entitled its questionnaire, "Effects of Waste Anesthetics on Health." With the exception of two Swedish studies (18,98) that validated their data with information from medical registries, the other studies relied solely on data collected from personal questionnaires. Neither of the Swedish studies revealed positive findings.

Male.—Infertility has been reported among men exposed to anesthetic gases; however, analysis of sperm number and morphology reveals no differences. Although experimental animals exposed to anesthetic gases appear to have normal reproductive function, alterations in sperm morphology have been observed in some studies (195). Reversible effects on spermatogenesis were reported when male rats inhaled nitrous oxide (260).

Female.—Although anesthetic agents have acute effects on the integrated control of the hypothalamic-pituitary-ovarian axis in women, the effect appears transient. Studies of exposure to halothane and nitrous oxide have been inconsistent with respect to fertility effects in females and embryolethality and fetotoxicity effects on the embryo/fetus. Nitrous oxide does not destroy oocytes in rodents (147).

Pregnancy.—Although studies are somewhat inconsistent, exposure to anesthetic gases has been correlated with increased rates of spontaneous abortion (147,346). Women working as dental operatory chairside assistants show increased rates of spontaneous abortion compared with wives of operating room personnel and wives of dentists (147). Experimental animals exposed to various anesthetic agents (227) demonstrate delayed development. Analysis of infant outcome in cases of either maternal or paternal exposure has been inconsistent with respect to congenital malformations in humans (147).

Epichlorohydrin

Epichlorohydrin, which is a liquid at room temperature, is a highly reactive compound used as an intermediate in the manufacture of a broad spectrum of chemicals, including agricultural chemicals, insecticides, coatings, adhesives, plasticizers, textile chemicals, and pharmaceuticals. An estimated 85,000 workers face potential exposure to epichlorohydrin (365).

Evidence suggests that epichlorohydrin is a potential human mutagen. Human somatic-cell chromosomal changes have been reported, both in vitro and in vivo (193,285,338).

Male.—In a study of testicular function in two cohorts of workers at two plants where epichlorohydrin was produced (236), semen of 128 of 216 eligible workers was compared with that of a 90-member control group. No differences were found between sperm count distributions in exposed workers and the control group. Further, no relationship was found between sperm count and either the duration or intensity of exposure to epichlorohydrin.

A 1980 study examined the fertility status of 64 men employed in the glycerin department of a Texas industrial chemical plant (376). Epichlorohydrin was one of three carbon compounds produced. The other two were allyl chloride and 1,3-dichloropropene. All of these are structurally related to DBCP, a pesticide known to cause sterility in male workers. Employees were divided into three subgroups on the basis of their work areas: 1) epichlorohydrin and allyl chloride, 2) allyl chloride and 1,3-dichloropropene, and 3) epichlorohydrin, allyl chloride and 1,3-dichloropropene. Employees were also classified by strength of exposure (a subjective measure) and duration of employment. No associations were shown between lowered fertility and exposure to epichlorohydrin, allyl chloride, or 1,3-dichloropropene when the 64 exposed and 63 unexposed employees were compared. Further, there were no differences between the three groups in measures of fertility (e.g., sperm count, percent viable sperm, sperm motility). A 1982 review found no studies that show an association between epichlorohydrin and human male sexual function (26).

The antifertility effects of epichlorohydrin on the male rat are well-documented. Reversible infertility in the absence of histologic damage to the gonads was first shown in male rats given epichlorohydrin orally at 15 mg/kg body weight for 12 days (26). Higher doses caused damage to the testes which resulted in permanent sterility. Exposure of male rats to 50 ppm epichlorohydrin by inhalation for 10 weeks resulted in infertility that was reversed 2 weeks after removal from exposure (170). At a lower exposure level of 25 ppm, fertility was impaired but not abolished in male rats. An exposure level of 5 ppm epichlorohydrin in air had no effect on fertility in male rats. In male rabbits exposed to 5, 25, or 50 ppm epichlorohydrin in air, no effect on fertility could be seen.

Female.—Among female rats inhaling 5, 25, or 50 ppm epichlorohydrin for 10 weeks prior to mating, no adverse effects were noted on the estrous cycle, pregnancy rate, or number and viability of the offspring (170). No studies of humans are available.

Pregnancy.—Although epichlorohydrin appears to have no specific adverse effects on the outcome of pregnancy in animals, there has been little study of possible effects. In pregnant rabbits inhaling epichlorohydrin at 2.5, 25, 50, or 100 ppm, no effects were observed in the absence of maternal toxicity (26). No significant effects were reported at up to 25 ppm for 7 hours/day on days 6 to 16 of gestation on pregnancy outcome in rabbits. No data are available for humans.

Ethylene Dibromide [EDB]

Ethylene dibromide is used chiefly as an antiknock additive in leaded gasoline. It was also used as a pesticide from 1948 to 1984, primarily as a preplanting soil fumigant against nematodes, but also to fumigate fruits, vegetables, grain, and grain-milling machinery. Pesticidal use of EDB is now limited to fumigation of citrus and tropical fruits for export and, until 1986, certain beehive equipment. EDB continues to be used as an intermediate in the synthesis of dyes and pharmaceuticals, and as a solvent for resins, gums, and waxes. It is used less frequently in fire extinguishers and as a catalyst in the synthesis of organic chemicals.

In 1983, an estimated 56,000 (66) to 108,000 (359) workers in the United States were potentially exposed to EDB during its production and use. Because most pesticidal use of EDB was halted in late 1984, these figures are now likely to be overestimates of current exposure. An additional 875,000 workers are potentially exposed to low concentrations of EDB while working with leaded gasoline. This use of EDB is declining as the demand for leaded fuel decreases (359).

A colorless, nonflammable liquid, EDB is absorbed into the body by skin contact and inhalation. It binds with many of the constituents of living cells, reacts chemically with and alters DNA, and can accumulate in body tissues over time with repeated exposures. Since it is similar in structure to DBCP, its potential mutagenic, carcinogenic, and male infertility effects have been investigated. Both continual and repeated intermittent exposures constitute a hazard to genetic mechanisms via accumulation of EDB in tissues (359). NIOSH recommends warning workers about the reproductive toxicity of EDB (244).

Male.—A 1979 study monitored fertility in wives of male workers in four plants who were exposed to EDB at levels up to 5 ppm (401). At three of the plants there was no evidence of fertility changes and at one there was a suggestion of lower fertility. Recent evaluation of workers exposed to EDB during its production suggests that exposure to levels below 5 ppm impairs spermatogenesis (350).

Adverse effects of EDB on the male gonads have been demonstrated in the rat and the bull. Atrophy of the testes and secondary sex organs occurred in rats inhaling 89 ppm EDB for 10 weeks (26). At this level of exposure, however, 20 percent of the animals died. At lower concentrations of EDB that were not significantly toxic (19 or 39 ppm), no specific effects on the gonads of male rats were seen. Calves and bulls were shown to be much more susceptible to a selective toxic action of EDB on the gonads. Daily oral doses of EDB averaging 2 mg/kg/body weight/day resulted in semen and sperm abnormalities and damage to the testes, which occurred in the absence of other signs of toxicity (26).

Female.—There are insufficient data to comment on the potential for adverse reproductive effects in women exposed to EDB. Chickens appear to be relatively sensitive to EDB as evidenced by impaired follicle growth and egg size. However, in one study, rat estrous cycles were affected only at doses that were lethal to 20 percent of the animals (26).

Pregnancy.—The effect of inhalation exposure of EDB during pregnancy was studied in rats and mice. In one series of experiments, pregnant rats and mice inhaled EDB at 20, 32, 38, or 80 ppm on days 6 to 15 of pregnancy (26). There was no apparent effect of EDB treatment on the incidence of major congenital malformations in the fetuses of rats or mice. Fetotoxicity was observed at doses that caused maternal toxicity. In one group of pregnant rats inhaling 32 ppm EDB, an increase in the incidence of minor congenital defects was observed in conjunction with slight maternal toxicity. In a 1983 study, rats were exposed to EDB at levels of 0.43, 6.67, or 66.67 ppm in air during pregnancy (333). Maternal toxicity was evident at the two higher dose levels, and the offspring showed signs of postnatal neurobehavioral impairment. No effects on the mother or fetus were evident from exposure to 0.43 ppm of EDB in air. EDB administered by daily intraperitoneal injection at 55 mg/kg body weight to pregnant rats on days 1 to 15 of gestation produced signs of maternal toxicity (significant change in maternal organ weights) but no evidence of fetotoxicity or teratogenicity (137).

EDB is a potent animal carcinogen and testicular toxin. Evidence indicates that human males are more susceptible than animals. Because data on fertility are equivocal, in late 1983 NIOSH began a cytogenetic and semen study of the effects of occupational exposure to EDB. Fifty workers exposed to EDB in the fumigation of fruit are under study, as are 50 nonexposed sugar refinery and plantation workers. Blood and sperm samples are being analyzed, and each participant has contributed a questionnaire covering demographic data, occupational history, and medical history (270).

Ethylene Oxide [EtO]

Ethylene oxide, a colorless gas, is a major industrial chemical ranked 26th in U.S. production of chemicals. The vast majority of EtO is found in chemical plants, where it is produced and used in the production of ethylene glycol for automotive antifreeze, polyester fibers and films, and detergents (368). EtO is also used in sterilizing equipment and supplies used in hospitals and health-care facilities, as a fumigant in the manufacture of medical products and foodstuffs, and in libraries and museums (107).

Because EtO is highly explosive and chemically reactive, the processing equipment containing it in chemical plants generally consists of tightly closed and highly automated systems. Such equipment is often located outdoors, and workers spend most of their shift in and around control rooms, away from the equipment. The greatest potential for worker exposure in these settings occurs during the loading or unloading of transport tanks, product-sampling procedures, and equipment maintenance and repair (368).

In contrast to chemical-manufacturing plants, health-care and medical-products industries use a very small portion of total EtO production, but workers in these industries face potentially high levels of occupational exposure to the chemical (368). Workers in hospitals and health care facilities are believed to be both the largest single group of workers exposed to EtO, and the group exposed to the highest levels of EtO (see table 4-1). Estimates of the number of workers exposed to EtO from all sources range from 100,000 (126) to 140,000 (271), including 75,000 health care workers employed in sterilization areas.

Exposure to EtO during sterilization of medical equipment is quite variable within a given hospital or health care facility, and also varies greatly from one hospital or health care facility to another. Some institutions may have several sterilization cycles per day, involving a number of different sterilization units. In other institutions, there may be only one sterilizer unit that is run infrequently. Other variables affecting exposure include:

- the nature and installation of the sterilization equipment,
- design and layout of the room housing the sterilizer,
- the nature and frequency of equipment maintenance activities,
- sterilizer operating practices, and
- the type and functional capacity of ventilation systems.

Exposures of sterilizer personnel to EtO consequently vary widely; some sterilizer personnel are exposed daily, and others may be exposed intermittently or infrequently (107).

Table 4-1.—Estimated Ethylene Oxide Fumigation Use and Potential Operator

Site	Ethylene oxide of operators (pounds × 10,000/year)	Estimated number
Manufacturing and production of sterile medical disposables	3.3-5.7	3,000-4,000
Hospitals (1976 figures)	822-1,000	11,000-26,000
Medical clinics	111	1,150
Dental clinics	65.5	400
Doctors, private	37	750
Dentists, private	7.3	80
Veterinarians, private and clinic (estimated)	0.1	NA[a]
Museums	0.7	15
Libraries and archives	1.9	40
Research laboratories:		
Animal breeding	50	25-30
Drug and medical device	550-900	NA[a]
Microbiological and cancer	5-25	NA[a]
USDA[b] high-containment research labs	4.3	10-15
USDA[b] APHIS[c] quarantine operations	0.7	200-300
Railroad cars	2	5-10
Beehives	1-2	30
Spices	750	60
Black walnuts	3.2	10
Cosmetics	24	25
Dairy packaging	32	30

[a] NA—Not available.
[b] USDA—United States Department of Agriculture.
[c] APHIS—Animal and Plant Health Inspection Service.

SOURCE: "Occupational Exposure to EtO, Final Standard," *Federal Register* 49(122):25734, June 22, 1984.

Major emissions of EtO into workroom air occur during discharge of EtO into floor drains, following opening of the door of the sterilization equipment after completion of a cycle, and during exchange of gas cylinders. Additional exposure may result from off-gassing of EtO from sterilized articles during aeration, leaks in the sterilizer system, and releases during maintenance of equipment. All of these variables hinder the determination of precise worker-exposure levels (107).

EtO is a recognized mutagen and has a genotoxic mode of action. At very low dose levels, (TWA of 1 to 10 ppm), mutagenic effects were observed (107). Changes in genetic material and alterations in DNA repair occur at average EtO exposure concentrations of 1 ppm. Effects observed in humans include unscheduled DNA synthesis, and deficiencies in DNA repair, sister chromatid exchange, and chromosomal aberrations, including quadriradials, a relatively rare aberration. These data demonstrate clearly the genetic toxicity of EtO in somatic cells and signal the potential of this chemical to damage germ cell DNA.

Male.—EtO has produced testicular damage and impaired fertility in rodents inhaling a toxic concentration (26). Guinea pigs inhaling 357 ppm EtO for 25 weeks showed general growth depression and testicular degeneration. Decreased fertility and dominant lethal effects were found in rats following a single 4-hour exposure to 1,000 ppm EtO in air. Exposure of male rats to 10, 33, or 100 ppm EtO in air for 12 weeks had no effects on fertility indices (336). A single intravenous injection of EtO at 25, 50, or 100 mg/kg body weight in male mice did not result in dominant lethal mutations when the animals were subsequently mated with untreated females (26).

Female.—A study of hospital workers using sterilization equipment revealed an increase in the spontaneous abortion rate that was correlated with exposure to EtO (143). Although some misclassification of the pregnancies according to exposure may have been possible, the data suggest a toxic effect of ethylene oxide on human reproduction (143).

Pregnancy.—Exposure of pregnant rats to 10, 33, or 100 ppm in air on days 6 to 15 of gestation resulted in fetotoxicity at the highest dose level, but no evidence of embryolethality or teratogenicity (335). Similar findings of fetotoxicity were reported in pregnant rats and rabbits inhaling 150 ppm EtO (138). The fertility of female rats exposed to 10, 33, or 100 ppm EtO in air, beginning 12 weeks before mating and continuing throughout pregnancy and lactation, was not affected although there were significantly fewer offspring born per litter in animals exposed to 100 ppm (335). Maternal toxicity did not result from the treatment, and survival and growth of offspring during the postnatal period were not adversely affected, even while the nursing mothers were exposed to EtO.

Formaldehyde

Formaldehyde is a colorless, flammable gas with a pungent odor. Formaldehyde may be used either in a water-based solution (i.e., formalin) or in solid form. In 1983, the United States used more than 7.5 billion pounds of formaldehyde in some 60 different industrial and laboratory applications (399). For example, formaldehyde and its derivatives are used: to give wet strength to paper; in transforming raw animal skin and fur into tanned leather; to harden and protect the gelatin surface of film and photographic papers; in textile processing; in the manufacture of particle board, plywood, and foam insulation; and as a preservative of biological material.

During a 1972-74 survey, NIOSH estimated that 1.6 million workers were exposed to formaldehyde. Of these workers, about 57,000 were exposed to formaldehyde for 4 or more hours per day. Nearly one-third of workers, some 507,200, were engaged in medical and other health services (367).

Formaldehyde is ubiquitous in the human environment and is a normal metabolite in human biochemistry. It is contained in cigarette smoke, car exhaust fumes, and in ambient air, even in remote areas. Formaldehyde can be found in a large variety of consumer products, ranging from permanent-press fabrics to cosmetics. The most common sources of exposure for the nonsmoking general population are particle board, plywood, and urea formaldehyde foam insulation. When new, these emit formaldehyde and can cause the levels in indoor air to become relatively high.

Male.—A 1984 study reported that formaldehyde exposure in men had no effect on sperm count or morphology (381). The human subjects in this study were 11 hospital autopsy service workers and 11 matched controls. Sperm counts were lower (but not significantly) in exposed men than controls, however, indicating the need for a larger study from which more definite conclusions can be drawn.

Data regarding the reproductive toxicity of formaldehyde in animals are limited. In male rats chronically exposed to formaldehyde at two doses (0.1 mg/liter in water, 0.4 ppm in air), no effects on fertility were seen (26). In a dominant lethal study treatment of male mice with single intraperitoneal injections of formaldehyde at 16 to 40 mg/kg body weight produced no effects on pregnancy rate or dominant lethal effects (25).

Female.—A study of 446 Soviet workers exposed to urea formaldehyde resins in a fabric plant found menstrual disorders in 47.5 percent of exposed fabric finishers and inspectors. By contrast, only 18.6 percent of the 200 industrial saleswomen in a comparison group were found to have such disorders. Dysmenorrhea was the most common disorder reported. No test for statistical significance was performed, but the highest frequency of menstrual disorders occurred among the youngest women, and among the fabric finishers who experienced the greatest exposure. Formaldehyde concentrations ranged from less than 0.05 ppm to 3.7 ppm, depending on the area of production (329). A 1980 study found that gynecological disorders accounted for only 2.3 percent of all disorders in 13,000 cases of unfitness for work at a plywood factory where women were exposed to formaldehyde (15). Another 1980 study reported no increase in miscarriages among women exposed to formaldehyde in the home

Table 4-2.—Workplace and Ambient Exposure to Formaldehyde

Exposed population[a]	Number of individuals exposed
Industrial workers:	
Abrasives manufacturers	7,000
Particle board manufacturers	4,000
Resins manufacturers	6,025
Apparel manufacturers	777,000
High school biology students	3,834,000
Beginning medical students	16,000
Residents of new mobile homes	4,200,000
Residents of urban areas, exposed to ambient air	162,000,000

[a] Only a small sample of the various categories of workplace and ambient exposure is given.

SOURCE: Adapted from B. Hileman, "Formaldehyde: Assessing the Risk," *Environ. Sci. Technol.* 18(7):216A-221A, 1984.

(119). All of these studies are flawed by the fact that exposures were not measured. The study of Soviet workers appears to have confounding factors that prevent formaldehyde per se from being implicated as a reproductive hazard. There are no adequate studies of the effects of formaldehyde on female animal fertility or pregnancy.

Pregnancy.—In the Soviet study, anemia was the most frequent pregnancy complication in women exposed to formaldehyde (329). Although not analyzed for significance, this pregnancy complication was reported twice as often by the exposed group as by the unexposed group.

No difference in the frequency of spontaneous abortion was found in a comparison of pregnant women who sterilized medical instruments with formaldehyde and pregnant women not exposed to formaldehyde (143). Frequencies were based on total number of pregnancies, and rates were adjusted for age, parity, decade of pregnancy, smoking, and alcohol and coffee consumption. Of the children born to mothers exposed to formaldehyde, 17 percent weighed 2,500 to 2,990 grams. Only 11 percent of the babies born to unexposed women were in this borderline-low weight category. Whether variables know to affect birth weight were controlled is not known.

Pregnant mice given formaldehyde orally at doses up to 185 mg/kg body weight/day on days 6 to 15 of gestation showed no adverse effects other than maternal toxicity. Dogs who were fed diets containing 125 or 375 ppm of formaldehyde (corresponding to doses of 3.1 or 9.4 mg/kg/day) from days 4 to 56 after mating (26) showed no evidence of embryolethality or teratogenicity, although fetal weights were slightly reduced in comparison with untreated control animals. Postnatal development of pups from formaldehyde-treated mothers appeared to be normal, and the pups were reported to have subsequently produced normal litters. A more recent study showed no effect of formaldehyde on embryos when hamster dams were exposed on day 8, 9, 10, or 11 of gestation (278).

Rubber

The production of rubber involves an estimated 500 or more chemicals, including acrylonitrile, aromatic amines, 1,3-butadiene, carbon black, chloroprene, epichlorohydrin, mineral oils, nitroso-compounds, styrene and other solvents, and vinyl chloride. The reproductive toxicity of all of the individual chemicals involved, as well as various combinations of them, is poorly understood, although some are identified as reproductive toxins. The range of possible reproductive hazards caused by exposures in the rubber industry has not been comprehensively studied.

Researchers have not attempted to separate or to measure chemical exposures, although efforts have been made to identify specific work areas where greater exposures probably occur. Although accurate individual exposure estimates are difficult to make in an environment such as a rubber plant, evidence from reproductive as well as other studies suggests that the level of harm from chemical exposure may vary greatly throughout the plant, making such determinations important.

Information on reproductive and developmental effects is available for several of the chemicals involved in the production of rubber—chloroprene, 1-3 butadiene, and ethylene thiourea.

Chloroprene is a colorless liquid that is slightly soluble in water. It is used as a chemical intermediate in rubber manufacturing. Chloroprene at room temperature apparently dimerizes to several different compounds. It has been demonstrated that these reaction products are often more toxic than chloroprene, which may explain the inconclusive results obtained by several investigators. Since dimerization is likely to occur in industrial settings, the reproductive toxicity of the dimers may need to be explored in order to enhance understanding of the reproductive effects associated with chemical exposure in rubber plants.

1,3-butadiene is a gas, readily soluble in organic solvents, used in the manufacture of rubber, latexes, and resins. Although there are no data showing human reproductive effects of 1,3-butadiene, NIOSH recommended in 1984 that 1,3-butadiene be regarded as a potential occupational human reproductive hazard. The NIOSH recommendation was based on long-term animal studies that demonstrate maternal and fetal toxicity, teratogenicity, and testicular and ovarian atrophy (371).

Ethylene thiourea is a rubber accelerator, used to speed the curing process in the manufacture of rubber. It is available as a powder, or as a powder suspended in oil, which retards the dispersion of ethylene thiourea dust in the air. NIOSH recommended in 1978 that ethylene thiourea be handled as if it were a human teratogen. Based on data derived from animal studies, NIOSH found that ethylene thiourea poses a risk of teratogenesis, particularly to the central nervous system, that is greater than has been generally recognized. An estimated 3,500 workers in the rubber industry have potential occupational exposure to ethylene thiourea (365). A 1976 study of employees formerly exposed to ethylene thiourea (exposure ended in 1972), identified no increase in specific congenital anomalies such as hip dislocation, malformed trachea and esophagus, cleft palate, and heart disease among the offspring of exposed workers compared with those of nonexposed workers (332).

Male.—A Russian study found reduced sperm motility in workers after 6 years exposure to chloroprene and changes in morphology after 11 years (26,312). Few details of the study are given, so it is impossible to access the significance of the result. A threefold increase in the abortion rate in the wives of rubber workers was also reported. A NIOSH (1977) document reports sexual impotency with both loss of libido and sexual dynamics following exposure to high levels of chloroprene.

Female.—Menstrual disorders have been associated with chloroprene exposure (47 percent in exposed v. 10 percent in controls) (26). A 1976 study reported 6.1 percent sterility in chloroprene workers v. 2 percent in controls (312). Females appear to be less susceptible to gonadal toxicity than males (26,312). Fertility is not affected by chloroprene exposure in animals where the purity of the substance is known.

Pregnancy.—In 1983, two investigations focused on rates of spontaneous abortion and congenital malformations among women exposed to chemicals in the rubber industry. In one report (213), the rate of spontaneous abortion did not differ between pregnancies occurring during employment and those occurring before or after employment, after adjusting for differences in age. A case-control study of spontaneous abortion in the footwear department (a high-exposure area) of one plant indicated a tenfold increase in risk of spontaneous abortion for women exposed to rubber chemicals compared with unexposed women working in a nearby area of the plant. A second report (19) found an increase in pregnancy complications, including miscarriages and threatened abortions, among tire builders.

Exposure to pure chloroprene up to 25 ppm has no effect in animals. Following exposure to chloroprene where purity was in question, teratogenicity and embryo death were noted at concentrations as low as 1 ppm, suggesting that impurities or reaction products are responsible. Many of the chemicals used in the rubber industry are teratogenic in the chick embryo assay (186,187). Those with the highest teratogenic potential were the highly aromatic oils and tricresylphosphate.

Vinyl Halides

Vinyl halides are in widespread industrial use, especially in the manufacture of plastics. These chemicals are easily polymerized with acrylonitrile, vinyl acetate, and styrene to form pliable, lightweight plastics or resins. The best studied and most widely used vinyl halide is vinyl chloride, which may occur as a monomer or polymer, called polyvinyl chloride (PVC). Polyvinyl chloride occurs in a wide variety of commercial products, including clothing, upholstery, flooring, wire insulation, food containers, and phonograph records. Other vinyl halides of industrial importance are vinylidene chloride, vinyl bromide, vinyl fluoride, and vinylidene fluoride. Exposure to the vinyl chloride monomer, generally in the polymerization industry, is considered the most hazardous of vinyl halide exposures (171).

Studies of vinyl chloride provide exposure levels, at least on an industry-wide basis. However, the extent and type of exposure vary widely, according to the production facility and process utilized. Discrepancies among results may occur because of differences in exposure levels across studies and in the differences of exposures to other agents, such as organic solvents, during the production of vinyl chloride.

Male.—There is some evidence that vinyl chloride may cause sexual dysfunction in men (26). A study of pregnancy outcome among wives of 95 workers showed increased fetal loss following their husbands' exposure to vinyl chloride monomer. The greatest increase occurred in pregnancy outcome associated with husbands under age 30 (161).

The absence of dominant lethal effects in male rats and mice inhaling vinyl chloride has been demonstrated by high dose short-term exposure (30,000 ppm for 5 days), and lower dose subchronic exposures (5,000 ppm for 10 weeks or 1,000 ppm for 5 days). However, reduced mating performance and fertility have been observed in male rats inhaling 250 or 1,000 ppm for 11 weeks. Pregnant rats, rabbits, and mice exposed to vinyl chloride at concentrations up to 2,500 ppm have exhibited maternal toxicity and some embryolethality and fetotoxicity (26,149,169).

Pregnancy.—Vinyl chloride has also been associated with increased rates of fetal death following paternal exposure (161), and possibly associated with malformations of the fetal central nervous system following environmental exposure of both parents. Studies of female exposure have been limited and tend to focus on environmental rather than workplace exposure and to utilize aggregate rather than individual data.

Residents of Painesville, Ohio, the site of two PVC plants, showed a significant increase in central nervous system (CNS) malformation. Scientists from the Centers for Disease Control used Birth Defects Monitoring Program (BDMP) data to compare CNS malformations rates in Painesville and a similar Pennsylvania community housing a PVC plant with rates for both States (91). The study found no increase in CNS malformations in the Pennsylvania community, but did find an increase in the Painesville area, primarily in anencephaly and spina bifida. A small, follow-up, case-control study (cases = 15; controls = 30) failed to show an association with vinyl chloride exposure.

Table 4-3.—Workplace Vinyl Halide Exposures

Chemical	Estimated number of workers potentially exposed	
	Definite[a]	Probable[b]
Vinyl chloride	27,000	2,200,000
Vinyl bromide	360	26,000
Vinylidene chloride	6,500	58,000
Vinylidene fluoride	1,900	32,000
Vinyl fluoride	NA[c]	NA[c]

[a] Definite estimates are extrapolated from actual observations of the use of the specific chemical or the use of a trade name product known to contain the chemical.

[b] Probable estimates include additional extrapolations from observations of trade name products suspected of containing the chemical because of generic formulations.

[c] NA—not available.

SOURCE: U.S. Department of Health, Education, and Welfare, National Institute for Occupational Safety and Health, "Vinyl Halides Carcinogenicity," NIOSH/OSHA Current Intelligence Bulletin 28, DHEW (NIOSH) Pub. No. 79-102, Sept. 21, 1978.

BDMP data were also used to identify the rate of CNS defects in Kanawha County, West Virginia, which houses a polyvinyl chloride facility, as being higher than the national rate. In a follow-up, case control study, 46 cases with CNS defects were matched with 2 normal controls each. The study found no evidence that higher CNS rates in Kanawha County were related to parental exposure to vinyl chloride monomer (90).

Pregnant rats, rabbits, and mice have been exposed to vinyl chloride at concentrations of up to 2,500 ppm in air. A 1981 study reported that maternal toxicity, but not fetotoxicity or teratogenicity, resulted from exposure of pregnant mice to 50 ppm and exposure of pregnant rats and rabbits to 2,500 ppm vinyl chloride in air (169). Maternal toxicity, embryolethality, and fetotoxicity developed in pregnant mice exposed to 500 ppm vinyl chloride in air. Embryolethality in the rat was increased by inhalation of 1,500 ppm vinyl chloride early in pregnancy (days 1 to 9 of gestation) (26).

The mutagenicity of vinyl chloride raises concern for the integrity of germ cell DNA in exposed individuals. There is insufficient evidence to reach conclusions about fertility effects in animal reproduction.

Hormones

Synthetic hormones have a wide variety of uses, ranging from supplements in animal feeds to human pharmaceuticals (e.g., oral contraceptives, cancer therapeutic agents). Occupational exposure to synthetic hormones occurs chiefly during their production in pharmaceutical plants. The principal exposure of workers is usually to the synthetic estrogens ethinyl estradiol and diethylstilbestrol (DES) or to synthetic progestogens. Sources of exposure are via the air and direct contact, especially when hygienic or prophylactic measures are neglected. In the United States, an estimated 3,000 persons are exposed to ethinyl estradiol in the work environment (140).

There have been few studies of the reproductive effects of workplace exposure to synthetic hormones. Despite their small number, however, studies of these and other hormones in clinical settings provide a broad data base for evaluation and identification of site and mechanism of action. The literature is limited to data on observations in factories producing oral contraceptives and synthetic estrogens. These studies are noteworthy for their: 1) efforts to measure workplace exposure levels of the hormones, 2) measurement of exogenous hormones in the worker's bloodstream as exposure indicators, and 3) focus on exposure of both male and female workers.

Certain methodological problems (e.g., difficulty in measuring the clinical effects of exposure) complicate studies of this type. Effects are both subjective (e.g., complaints of loss of libido), and difficult to quantitate (e.g., gynecomastia). Clinical examination is not always conclusive; for example, 30 percent of the nonexposed adult male population may present with gynecomastia (139). Uncertainty also exists in identifying the most appropriate indicators of exposure and outcome. Despite these difficulties, adverse reproductive effects reported following occupational exposure to hormones are consistent with the well-defined biological actions of these compounds.

Male.—A 1984 study (237) of 22 hormone-exposed men found an increased incidence of breast swelling, tenderness, and lumps or nodules, and decreased total blood estrogen levels, but no detectable evidence of synthetic estrogens in the blood. These changes are consistent with occupational exposure to and absorption of synthetic estrogens.

Female.—Lower average total blood estrogen levels have been reported in hormone-exposed female workers (237). Again, none of the women had detectable evidence of synthetic hormones in their blood. Among 24 female employees exposed to the synthetic hormones mestranol and norethindrone, 50 percent experienced intermenstrual bleeding, compared with 17 percent of a group of 60 nonexposed women (140).

Pregnancy.—The adverse reproductive effects of the synthetic estrogen diethylstilbestrol (DES) have been well-documented in pregnant mice, rats, hamsters, rabbits, monkeys, and humans (260). In pregnant mice, daily subcutaneous injections of DES at doses ranging from 0.01 to 10 mg/kg body weight/day during gestation caused severe developmental and functional disturbances in both male and female offspring. Females exhibited decreased fertility, sterility, and abnormalities of the genital tract; male offspring showed growth inhibition, sterility, and alterations of the reproductive tract. Similar effects were observed in the offspring of rats and hamsters treated with DES during pregnancy. Abnormalities of the genital tract were reported in female offspring of monkeys given DES orally at doses of 1 mg/day from day 21, 100, or 130 of gestation to delivery. Women exposed to DES in utero have been demonstrated to have abnormalities in the development of the uterus and cervix. In addition, DES is a transplacental carcinogen in women and experimental animals.

High levels of corticosteroid hormones in early fetal life have been associated with developmental toxicity in animals. Hydrocortisone acetate, a synthetic glucocorticoid hormone, has been studied for its ability to induce renal anomalies in the offspring of pregnant rats given an injection of 250 mg/kg body weight during the gestation period of fetal organ development. Polycystic kidney disease may also be induced by injecting newborn rats, rabbits, hamsters, and mice with the hormone because kidney development continues postnatally in these species.

Although workplace exposure to hormones such as DES and hydrocortisone acetate is primarily through inhalation and most laboratory studies have administered the hormones in feed and through injections (77,260), these differences do not obscure the clear reproductive toxicity that follows occupational exposure to hormones.

Undefined Industrial Exposures

A number of studies have examined the effects of particular occupations on workers' reproductive function. These studies do not specify the individual chemicals to which the workers are exposed, nor do they attempt to quantify exposure.

Agricultural Work.—A 1973 study (408) examined white-blood-cell cultures from 42 pesticide-application workers and 16 nonexposed workers to evaluate chromosomal characteristics. Increases in frequency of chromosomal abnormalities, especially in workers with heavy herbicide exposure, occurred during heavy-spraying seasons.

A 1978 study of five Israeli insecticide workers found impaired spermatogenesis, chromosomal breakage, and Y-chromosome damage. The five men, who were infertile, had been frequently exposed to various chlorinated and phosphate organic insecticides (324).

A series of case reports reported impotence among four of five farm workers exposed to unspecified chemicals. The impotence was not accompanied by a decrease in libido. When contact with the chemicals was stopped and hormone therapy given, the four workers recovered sexual function (101).

Laboratory Work.—A 1977 study (116) found an excess of chromosomal abnormalities in the white blood cells of 73 workers in laboratories and in the printing industry. An increase in chromosomal abnormalities was found in 14 children of 11 women who had worked in laboratories while pregnant.

A study of pregnancy outcome among 32 women working in a Swedish hospital laboratory found an increased risk of spontaneous abortion, which occurred in 17 of 71 pregnancies, when pregnancy occurred in conjunction with laboratory work. This study was conducted on a relatively small population, and confounding variables were not factored into the analysis (341).

A 1979 study (23) of the relationship between delivery outcome and women working in medical professions covered 1,500 women working in hospitals from 1965 to 1975 who gave birth during the period. The hospital workers exhibited increased rates of cesarean deliveries and threatened abortions, and during 1 year of the study, perinatal death.

A 1984 report examined delivery outcomes of 1,161 infants born to Swedish laboratory workers and compared them with the total number (98,354) of births in Sweden in 1976. Although an increase in perinatal deaths and congenital malformations was found among infants of a subset of the laboratory workers, no specific type of laboratory or laboratory worker was found to be associated with these outcomes (97).

Two other Swedish studies have found that laboratory workers are more likely to give birth to infants with congenital malformations of the gastrointestinal tract. A 1979 study (230) looked at perinatal death and malformation rates in 322 deliveries to women working at a Swedish university during their pregnancies. Of these women, 245 were laboratory workers while pregnant. No occupational effect on perinatal deaths was observed, but the study did show an increased rate of congenital malformations among offspring of laboratory workers. Gastrointestinal defects appeared to be especially elevated. A 1982 study of this outcome among pregnant women laboratory workers (99) found that infants with gastrointestinal atresia were more likely than normal infants to have mothers who were laboratory workers.

Oil, Chemical, and Atomic Work.—A 1984 survey of reproductive hazards among 1,280 male oil, chemical, and atomic workers exposed to halogenated hydrocarbons (315) in 7 U.S. plants was conducted by postal questionnaire. Workers in these plants used the chemicals ethylene dichloride, methyl chloride, vinyl chloride monomer, chlordane, epichlorohydrin, and perchlorethylene. Oil, chemical, and atomic workers not exposed to any brominated or chlorinated hydrocarbons served as a comparison group. Subjects were placed, on the basis of occupation, in "higher," "lower," or "no-exposure" categories.

The salient finding of this industrial study was an increase in infant deaths among the offspring of exposed male workers. The rate was 2.3 and 4.6 times greater for the "lower" and "higher" exposure workers, respectively, than for the nonexposed workers.

Pulp and Paper Work.—A study of female employees in the Swedish pulp and paper industry examined congenital anomalies and perinatal survival from 1973 to 1977 (38). Information on all births was gathered from the Swedish Medical

Birth Register. The number of congenital malformations, based on 890 deliveries, was close to the Swedish norm. When pregnancy outcomes were divided into specific job categories of the mother, the highest frequency of birth defects (4.0 percent) and perinatal deaths (1.8 percent) occurred among women in the "converting" section, where paper is refined into various products. Some of these workers were listed as having exposure to ethylene acetate, glues, and various stains.

Textile Work.—Medical records and data from questionnaires in Denmark indicate that female textile workers exposed to textile dyes experienced a fivefold increase in risk of infertility when these data were adjusted for age, education, residence, and parity. The risk of infertility among textile workers was greater than for women working with cutting oils, dry-cleaning chemicals, lead, cadmium, or mercury. No exposure levels were provided (293).

Several studies have examined the frequency of spontaneous abortion among women in the textile industry, although none of these studies, which are generally part of larger industrial investigations, focuses solely on this industry. In a 1977 Iranian study, the rate of spontaneous abortion (12 percent) was greater among textile workers than among nonworking women (175). More than 70 percent of the women interviewed were at least 30 years of age and had been employed in one of two local factories for more than 15 years. No specific workplace hazards were cited in the report.

A more recent investigation of spontaneous abortion among women in textile industries yielded similar findings (146). Unlike the Iranian study, this investigation took the husband's occupation into account. Hospital discharge data were employed to obtain information on the study group and their families in the community of Kokkola, Finland. While women in the town worked mainly in the textile industry, men were employed in the metal, leather, and chemical industries. The highest rate of spontaneous abortion in Kokkola (12.2 percent) was recorded among women textile workers. This rate was significantly higher than for women who did not work outside the home (6.3 percent), but only slightly higher than the rate for other economically active women (11.4 percent). A subgroup of women working as seamstresses in the textile factory had a spontaneous abortion rate of 20.4 percent. When the husband's occupation was also considered, women employed in textiles married to men employed at the metallurgical factory had a rate of spontaneous abortion of 16.0 percent. The authors suggest that higher rates of spontaneous abortion among the combined occupations may be due in part to a paternal effect. Although the husbands' jobs in the metallurgic factories were unspecified, possible exposures to arsenic, zinc, cobalt, sulfur dioxide, hydrogen sulfide, and cadmium were suggested.

A Swedish study found an increased rate of spontaneous abortion among both women and wives of men working in rayon textile jobs. The investigators noted that viscose rayon industries use hydrogen sulfide and carbon disulfide. No actual exposure data were provided (144). These studies suggest that occupational exposures during pregnancy in the textile industry are associated with an increased risk for female infertility and spontaneous abortion.

EFFECTS OF WORKPLACE PHYSICAL AGENTS ON REPRODUCTIVE FUNCTION

Workers in every occupational field are exposed to one or more physical agents in their workplace environment. The variety of forces encompassed by the term physical agents includes such natural forces as radiation, atmospheric pressure, and electric, magnetic, and gravitational fields. It is essential to recognize the close relationship between physical agents in the occupational environment and these same agents as integral parts of the natural environment. With few exceptions, these physical energies are, in fact, elemental forces that have shaped the evolution of life on earth. The form, behavior, and function—including reproduction—of human, monkey, mouse, rat, and dog developed under the influences of natural gamma rays, ultraviolet light, gravity, varying barometric pressures, and hot and cold temperatures.

As important as natural physical agents have been from an ecological perspective, they do not become notable agents of biological stress until: 1) above-normal levels are created artificially in industrial and commercial environments, or 2) the background levels become abnormal. The physical factors that have most often been considered as potential occupational hazards include ionizing radiation, optical radiation, radiofrequency/microwave radiation, electric and magnetic fields, atmospheric pressure, hot or cold environments, noise, and vibration.

Certain health effects resulting from occupational exposure to physical forces, such as noise-induced hearing loss, heat stress, and vibration-induced numbness, have been recognized for decades. Unfortunately, very few well-documented studies have been conducted for the specific purpose of evaluating the reproductive effects of exposure to physical forces in the workplace. Data on the adverse effects on reproduction from occupational exposure to physical forces are therefore in most cases either inferential or nonexistent.

Ionizing Radiation

Ionizing radiation is energy that is transmitted in wave or particle form and is capable of causing ionization (ejecting orbital electrons) of atoms or molecules in the irradiated tissue. Alpha particles and beta particles are forms of ionizing radiation that interact directly with irradiated tissues to cause ionization, whereas gamma and X-rays are forms of electromagnetic radiation that generate secondary particles in the irradiated tissues which subsequently lead to ionization. Reactors and high-energy accelerators produce, in addition to gamma and X-rays, protons, neutrons, and other particles that are effective in producing tissue ionization either directly (protons) or indirectly (neutrons).

The critical element for defining the biological effect of ionizing radiation is energy deposition (i.e., absorbed dose), since the different types of ionizing radiation vary in their penetrative powers and number of ions produced. The unit used to quantify the energy deposited in matter by ionizing radiation is the rad, defined as 0.01 joules per kilogram of irradiated material. Since different types of radiation can deposit the same total energy but produce different amounts of damage, a different unit, the rem, is used to quantify the degree of biological damage. Rems are defined as a factor Q times rads, where Q is set equal to 1 for gamma and X-rays, and 20 for alpha particles. Thus, at equivalent energy depositions, the alpha particle will produce 20 times the biological damage of gamma and X-rays. The currently recommended limit for workers exposed to ionizing radiation, set by the Federal Radiation Council (FRC, 1960) and incorporated into regulatory limits by most Federal agencies (e.g., NRC, 1977) is 3 rems/quarter (3 months) for the whole body, or head and trunk, lens of the eyes, gonads, or blood-forming organs. This limit is subject to the further constraint of a cumulative lifetime limit expressed as $5(N-18)$ rems, where N is equal to the worker's age in years. Some Federal agencies (e.g., the Departments of Defense and Energy) use a simpler, more restrictive limit of 5 rems/year.

A major source of human exposure to ionizing radiation is natural background radiation. The two sources of this exposure are cosmic radiation produced by collisions of high-energy particles impinging on the earth's atmosphere, and the radioactive elements (radionuclides; e.g., radon, potassium-40) commonly found in soil, brick, concrete, and stone. The total whole-body dose due to natural sources averages about 100 millirems per year; the dose to the lungs from natural sources is about 500 millirems per year; and the average gonadal dose from natural radiation is about 80 millirems per year (250,298). Added to this exposure from background radiation is the dose received from medical use of X-rays, which contributes about 20 millirems per year to gonadal exposure. Other minor sources of nonnatural exposure are atmospheric weapons testing, nuclear powerplant operation, consumer products, and building materials. Tobacco smoking may also result in substantial localized radiation exposures to points within the respiratory tract, possibly reaching 8,000 millirems per year (250).

Occupational Exposure to Ionizing Radiation

Some 1.32 million persons are presently occupationally exposed to ionizing radiation each year. About 44 percent of all exposed workers are employed in medicine, 23 percent in industry, 16 percent in government, and 11 percent in the nuclear fuel cycle. Workers in the nuclear fuel cycle accounted for the largest share of the collective dose (37 percent), followed closely by those in medicine (27 percent), and industry (25 percent) (see chapter 7). Comprehensive surveys of the numbers of workers exposed and their doses, age, and sex distributions have been published by EPA (372,373). In general, the exposures are low. However, it is important to remember that ionizing radiation causes dose-related damage to all tissues.

Industrial use of ionizing radiation is now rapidly expanding, both in terms of its application to industrial processes and the type of industry involved. Future developments in the industrial application of ionizing radiation are likely to be focused in the area of radiation processing. Research is being conducted on radiation processing to achieve cross-linking, polymerization, grafting, and free-radical generation in the chemical industry, and in the production of flooring, furniture, textiles, adhesives, paints, membranes, and wood/plastic composites (42).

Preservation and sterilization of foods, spices, cosmetics, and pharmaceuticals by irradiation is also rapidly approaching large-scale commercial application (42,288). These efforts will, of necessity, expand because of the ban on ethylene dibromide for similar uses. The radiation source used in sterilization can be either machine-generated electrons or gamma rays from cobalt-60 or cesium-137 (288). Reduction of microbial load and improvement in food properties occur with applications of about 100,000 to 1 million rads, and sterilization for commercial purposes requires about 1 million to 5 million rads.

Concern for worker exposures occurring during radiation-processing operations is greater than for other industrial or medical applications. Problems can be foreseen due to the experimental nature of the processes, the high doses of radiation employed, the likelihood that radiation processing will be conducted in small establishments with limited resources for protective measures, the lack of employee training regarding the hazards involved, and the absence of regulatory standards and guidelines for controlling exposures. No information currently exists to indicate the magnitude of potential exposure of men and women engaged in these newly emerging occupational tasks (288). This is therefore a research area of major concern because ionizing radiation is known to exert profound effects on the developing embryo/fetus and child and on reproductive function in men and women.

Male.—Ionizing radiation produces dose-related impairment of testicular function. There is some indirect evidence that occupational exposure to radiation is associated with diminished sex drive and decreased sperm viability in men (339). High doses of ionizing radiation clearly have an adverse effect on the gonads of men. Although the effects of relatively low doses of ionizing radiation on male reproductive function (below 5 to 10 rads) are not well understood, sperm production is suppressed by doses of X-irradiation as low as 15 rads (71). Sperm production is transiently eliminated with doses of 50 rads. At high dosages, in the range of 236 to 365 rads, severe spermatozoa damage occurs which persists for many months (75). Radiation doses greater than 400 rads are associated with the complete cessation of testicular function. Although it occurs rarely, recovery of sperm production is possible, even following dosages as high as 400 rads. There are numerous case reports of testicular damage produced by radiation therapy for malignancies (325), but well-documented reports on the effects of occupational exposures are limited.

Testicular gamma and X-irradiation in animals exert profound effects on developing sperm. Numerous studies have been conducted in mice to assess dose-response relationships for induction of sperm abnormalities (48,269). When mice were exposed to testicular X-irradiation, the dose to produce a doubling in the number of abnormal sperm in comparison with controls was determined to be 39 rads (409). A 1983 study determined that the dose of X-irradiation to produce a 50 percent suppression of type A spermatogonia was 30 rads for the mouse and 917 rads for the human (71). This indicates that human type A spermatogonia are about 3.1 times more sensitive to ionizing radiation than are mouse spermatogonia. Irradiation of the testes also has a mutagenic effect on male germ cells, as evidenced by reduction in post-implantation survival of the offspring of exposed male (313) animals.

Female.—In the female, the reproductive process is susceptible to radiation-induced damage in several ways. Because females are born with a fixed supply of oocytes (egg cells), damaged egg cells cannot be replaced (see chapter 3). Exposure of these cells to ionizing radiation, either during gestation or following birth, can cause reproductive disorders at puberty and during reproductive life. There is evidence that exposure during childhood may lead to disorders of the endocrine system, which subsequently give rise to infertility or failure to undergo normal pubertal development.

Animal studies demonstrate similar effects with a dose-related impairment of reproductive processes. All tissues of the reproductive tract are susceptible to the adverse effects of ionizing radiation but exhibit different dose-response curves. Numerous studies of the effects of ionizing radiation on ovaries, oocytes, and reproduction have been conducted in rodents, primates, and many other species (21). The vast body of data from animal studies reveals wide variations in susceptibility according to species, age, egg-cell stage, and follicle size (22,221). For example, extreme sensitivity of female egg cells to ionizing radiation is seen in postnatal mice and in prenatal squirrel monkeys. Oocytes in women and in adult rhesus monkeys, by contrast, appear to be relatively resistant. In sensitive animals, such as the juvenile mouse, destruction of immature oocytes can result from dosages of less than 6 rads.

Pregnancy.—Exposure of pregnant women to levels of greater than 20 rads leads to birth defects, while lower exposures in the region of 1 to 10 rads are associated with increased mental retardation and childhood leukemia and other cancers in their offspring (218,251). The National Council on Radiation Protection and Measurements (252) recommends that workplace exposure of a fertile woman be controlled to ensure that if she becomes pregnant her fetus will receive a cumulative exposure of no more than 0.5 rads.

Understanding of the teratogenic effects of ionizing radiation on fetal development dates to the explosion of the first nuclear weapon in 1945. Extensive retrospective epidemiological surveys were conducted on individuals exposed to radiation in utero in Hiroshima and Nagasaki (44,277, 343,402,403,405,406). These studies, coupled with earlier case reports, provide clear evidence of severe teratogenic effects, particularly the occurrence of microcephaly (reduced size of the brain), and severe mental retardation.

The effects of exposure on reproductive function are not known for the low-dose range in females although clinical data suggest that reproduction is not impaired. The evidence for harmful effects at high doses is clear, however. High doses can cause sterility and initiate menopause. Some effects of chromosomal abnormalities have been observed in women who were exposed to ionizing radiation prior to pregnancy, but important confounding variables may have biased results, and dosages were unknown.

Nonionizing Radiation

The term nonionizing radiation refers to the region of the electromagnetic spectrum where the energy of the emitted photon is incapable of ionizing atoms or molecules in the irradiated tissue. The lower wavelength limit for nonionizing radiation is considered to be 100 nanometers [nm], which corresponds to ultraviolet light. Succeeding portions of the spectrum correspond to visible light (400 to 750 nm wavelength), infrared radiation (0.75 micrometers [mm] to 750 nm wavelength), and radiofrequency radiation (1 millimeter [nm] to 10,000 kilometers [km] wavelength). As wavelength increases along the electromagnetic spectrum, wave frequency decreases. Considerable confusion arises from the fact that the anxiety-provoking term "radiation" is applied to X-rays (i.e., ionizing radiation) as well as to microwaves, radio and television transmission signals, and other forms of nonionizing energy. These forms of energy are in fact significantly different with respect to biological activity. All humans are under constant exposure to natural or man-made sources of nonionizing radiation, thereby complicating the design of any population study to assess the health effects of occupational exposure.

Ultraviolet Radiation

Ultraviolet radiation is produced naturally by the sun, and artificially by arcs operating at high temperature. Exposure to ultraviolet radiation in the workplace is associated with incandescent, fluorescent, and discharge-type light sources, as well as with welding and cutting torches, electric arc furnaces, plasma torches, and lasers. In addition, outdoor workers, such as farmers, fishermen, lifeguards, and construction workers receive substantial solar exposures. Ultraviolet radiation can be expected to occur in all occupations involving germicidal lamps, welding arcs, and plasma torches, and in industrial drying and curing processes, printing processes, and chemical manufacturing operations.

Visible Light

Visible light is provided by the sun and by artificial light sources. Industrial exposure to visible light is additionally associated with highly incandescent lights and various types of arc processes. Many sources of high-intensity visible light also produce substantial thermal energy.

Infrared Radiation

All objects emit infrared radiation, which increases as a function of temperature. The sun is a major source of infrared radiation. Occupational exposure occurs either directly from lamps or indirectly from heat sources. The most widely recognized industrial exposures to infrared radiation are from hot furnaces, molten metals or glass, and arc processes.

Laser Radiation

A laser (acronym for "light amplification by stimulated emission of radiation") operates in the infrared, visible, and ultraviolet regions of the electromagnetic spectrum. Lasers are sources of monochromatic optical-frequency waves, whose output can be focused to form extremely high-power beams (127). The source of laser radiation can be a solid, a liquid, or a gas that can be made to fluoresce. Sources in use include ruby, neodymium, helium, neon, argon, krypton, carbon dioxide, and an yttrium-aluminum-garnet combination.

The laser has been of great value in numerous segments of industry, and its applications continue to expand. In the biomedical field, lasers are used in the detection of tumors, to measure circulation and components of blood, and as optical knives to perform delicate surgery. Several methods have recently been developed in which lasers are used to detect air pollutants with great specificity and sensitivity. Lasers are used in metal-working and in the aircraft industry to drill holes, particularly on curved surfaces, with great accuracy and precision. A recent development in laser applications is in communications and information transfer with fiber optics.

The harmful effects of optical radiation appear to be restricted to the surface of the body, especially the skin and eyes. Lasers operating in the visible or near infrared wavelength regions may produce severe retinal burns of the eye, and lasers operating in the infrared region (e.g., carbon dioxide lasers) may produce surface burns on the cornea. Damage is primarily the result of tissue-heating, which causes protein destruction (denaturation) and the typical symptoms associated with burns. An additional biological effect of ultraviolet and infrared radiation, and of lasers, is excitation of intracellular organelles unrelated to tissue-heating (81). The health effects resulting from thermal excitation of cell organelles are not understood. Ultraviolet radiation is also regarded as a cause of skin cancer. There are no known reproductive effects in humans and lower animals associated with occupational or environmental exposure to optical radiation. In many animals, changes in the ambient levels of light are a powerful modulator of reproductive behavior.

Radiofrequency/Microwave Radiation

The applications of these man-made electromagnetic fields are extremely diverse and rapidly expanding. In terms of potential health effects, two frequency ranges are receiving focused attention. One is the microwave and shortwave frequency range (several MHz to 100 gigahertz (GHz) used by the military and for communications. The other is the extremely low-frequency range (10 to 60 Hz) associated with high-voltage power lines. There is no question that the thermal effects of radiofrequency and microwave radiation are hazardous. There is, however, little agreement as to the potential for health hazard produced by the nonthermal effects of this physical force.

For a human, significant heating will not occur with radiofrequency radiation having a frequency below 15 MHz and a wavelength greater than about 20 m (i.e., television and radio transmission, radiation from power lines). The electromagnetic radiation used in radar is in the microwave frequency range capable of inducing thermal and subthermal biologic effects in humans (234). The American National Standards Institute (ANSI) Committee C95 has recently proposed revised guidelines (8) for safe exposure to radiofrequency electromagnetic fields which acknowledge that prolonged whole-body exposure at intensities above 100 mW/cm^2 are dangerous at frequencies at which significant energy is delivered to the human body. In humans, the radiation absorption efficiency reaches a maximum at a frequency of 77 MHz for a person 1.75 m tall who weighs 70 kg (117). The majority of industrial radiofrequency sources operate from 10 to 40 MHz, whereas a microwave oven operates at 2,450 MHz. Diathermy electromagnetic waves (27.5 Hz) have great penetration into the human body and produce significant heating, while microwaves with frequencies above 10,000 MHz have little penetration (44).

Workplaces designated as hazardous due to the presence of radiofrequency/microwave radiation are generally associated with antenna systems, emitters, generator tubes, and other high-frequency units. The adverse health effects of exposure to radiofrequency/microwave radiation that result in tissue heating are well-documented (364). The health effects of subthermal doses remain unclear, particularly with respect to low-frequency and weak-field radiation.

Male.—The only form of nonionizing radiation that has been repeatedly associated with damage to male gonads is radiofrequency/microwave radiation. The available evidence is incomplete, however, with respect to dosage and influence of other variables. There is little doubt that radiofrequency/ microwave radiation of sufficient intensity can damage the testes by thermal action. Most studies of occupational exposure to radiofrequency/microwave radiation have involved military personnel. Clinical studies of radar operators in the U.S. Navy showed no adverse effects on male fertility.

Numerous studies have been conducted on testicular and reproductive function in rats and mice exposed to radiofrequency/microwave radiation. Testicular degeneration is clearly associated with microwave dosages sufficient to cause tissue heating (75,203,314). At a dosage of microwave radiation (1.3 GHz) sufficient to cause a net change in body temperature of 1.5° C, no effects were seen on the testes of rats (203). In contrast to the evidence for effects of ionizing radiation, evidence concerning a mutagenic effect for microwave radiation is inconsistent and conflicting (313). Yet impaired male fertility as evidenced by a reduced pregnancy rate in mated females can be achieved with sufficient dosages of microwaves (189). The extent to which thermal effects account for these results is not clearly established.

Female.—Epidemiological studies of microwave workers and military personnel exposed to radar have not provided clear evidence for the development of pathologic damage, reproductive failure in women, or malignancies (233). These negative and in some cases equivocal results may reflect inadequacies in the studies (e.g., inadequate dose information, inappropriate control groups, and lack of recognition of concomitant exposure to toxic agents). There is thus a need for well-designed and carefully controlled epidemiological studies of workers and other populations exposed to measured amounts of radiofrequency/ microwave radiation. The presently available data suggest that the adverse effects of radiofrequency /microwave exposure are primarily, if not exclusively, the result of tissue-heating. Occupational exposure of women to radiofrequency/microwave radiation at typical power densities would not be expected to produce sufficient internal tissue-heating to harm the fetus or the reproductive organs.

Pregnancy.—The adverse effects of prenatal exposure to radiofrequency/microwave radiation at various frequencies have been extensively studied in rats, mice, chickens, Japanese quail, and insects. Studies have been concerned with morphologic alterations, as well as more subtle neurobehavioral changes. The power levels employed in many of these studies were sufficient to indicate that fetal malformations may have resulted from hyperthermia. At lower power densities, there appears to be a minimum threshold level for induction of fetal abnormalities. Exposures of rats to a power density of 35 mW/cm² GHz continuous-wave microwave radiation on gestation days 1 to 6 produced a decrease in implantation sites per litter and decreased fetal weight (254). Exposure to a power density of 30 mW/cm² on days 6 to 15 of gestation produced a slight increase in fetal malformations. No effects on the offspring were observed when pregnant mice were exposed to power densities of 5 and 21 mW/cm².

Negative results have also been obtained with pregnant rats exposed to 915 MHz microwaves at a power level of 10 mW/cm² (166), and with pregnant rats exposed to 100 MHz radiation (the frequency region of maximum human absorption) at a power density of 25 mW/cm² (198,199). These exposures produce no increase in maternal temperature. It therefore appears that a threshold for induction of teratogenic effects in mice and rats by radiofrequency/microwave radiation may be in the power density region of about 30 mW/cm². These results also suggest that the 1982 ANSI exposure standard of 1 mW/cm² for frequencies between 30 and 300 MHz will provide adequate protection of pregnant women and the human embryo/fetus. It is important to note, however, that there is a considerable body of disagreement concerning the nonthermal effects of nonionizing radiation. Additional study of these effects will be necessary before acceptable exposure levels can be established.

Ultrasound

Ultrasound is a mechanical vibration of an elastic medium having a frequency range beyond 16,000 to 20,000 Hz, which is above audible frequency for the human ear. Low-frequency ultrasound (18,000 to 30,000 Hz) of high intensity (6 to 7 W/cm²) is widely used in industry in cleaning baths for metal and fabricated parts; in welding, brazing, and soldering; for electrolytic coating; and for acceleration of chemical reactions. Low-frequency ultrasound is also a compound of the noise produced by jet engines, gas turbines, and powerful pneumatic devices.

High-frequency ultrasound is more readily absorbed by the surrounding medium and does not travel in air. Penetration of human tissue by ultrasound decreases as the frequency increases. High-frequency ultrasound (500 kHz to 5 MHz) of low intensity (0.1 to 10 W/cm²) is widely used for detection of flaws and structural analysis of matter.

The medical applications of ultrasound have greatly increased in recent years, particularly in obstetrical diagnostic procedures (211). Two types of ultrasound are used with pregnant women. One is pulsed ultrasound, employing frequencies in the 1 to 10 MHz range with output intensities ranging from less than 1 to 10 mW/cm². The other is continuous-wave ultrasound, employing frequencies of about 2 MHz with output intensities ranging from less than 1 to 20 mW/cm². Continuous-wave ultrasound is used early in pregnancy for placental localization, confirmation of normal or abnormal pregnancy, detection of twins, and in monitoring fetal growth.

Worker exposure to ultrasound, particularly during the loading and unloading of parts of cleaning tanks, may result in damage to peripheral nerves and blood vessels of the fingers, hands, and forearms (306). The adverse effects in humans from high-frequency ultrasound are not clearly understood.

The teratogenic and embryotoxic effects of exposure to ultrasound have not been studied as extensively as those resulting from exposure to radiofrequency/microwave radiation. As with microwaves, when ultrasound exposure has been reported to induce fetal malformations, there is also an increase in maternal temperature (181). When pregnant mice were exposed to 1 MHz ultrasound at power densities up to 1.00 W/cm², no statistically significant effects on the fetus could be demonstrated (181). These results suggest that clinical applications of ultrasound diagnostic procedures in pregnant women at typical power levels below mW/cm² should not pose an unacceptable risk to the mother or fetus.

Video Display Terminals (VDTs)

Use of VDTs is rapidly expanding as a means to display alphanumeric information in the workplace. An estimated 5 million to 10 million VDTs were in use in the United States by 1980 (249). By 1990, it is projected that 25 million VDTs will be in use (13). The principal applications for VDTs are for data entry, data acquisition, interactive communication, word processing, computer programming, computer-assisted design, and computer-assisted manufacture. The expanding use of VDTs has created an area of special health concern with respect to workplaces and occupations that have been traditionally regarded as hazard-free. The major issue of concern is the potential for chronic worker exposure to radiation emitted by VDTs and its possible health-related consequences.

Most VDTs use cathode ray tubes, and in many respects are similar to television receivers. Cathode ray tubes emit visible radiation (light), but also emit ultraviolet and infrared radiation, and radiofrequency radiation in the 15 to 125 kHz frequency range. Cathode ray tubes also produce internal X-rays, which are effectively filtered by the tube face, thus preventing most emissions. Numerous field surveys and laboratory studies by industry, government, and independent groups have concluded that the emission of all types of radiation by VDTs is well within acceptable limits of exposure (13,249). It should be noted, however, that most VDT emissions are in the radiofrequency range below 300 kHz, where no enforceable emission standards have been established and adverse health effects are not well understood. A limit of 614 V/M or 100 mW/cm² for radiofrequencies between 10 kHz and 3 MHz is being recommended by the American Conference of Governmental Industrial Hygienists (5). This level is about 10 times higher than the VDT emissions in the 10 kHz-100 MHz range measured under worst-case conditions in a study by the Center for Devices Radiological Health (249). In the same study, no X-ray emissions could be detected from 91 VDT units operated under normal conditions.

Reports of clusters of spontaneous abortions, miscarriages, and birth defects among VDT operators have raised serious concerns over safety. Although at least two of these clusters have been investigated, no association has been confirmed for VDT work and increased risk for adverse reproductive outcome (249). The only documented causal role of VDTs in inducing birth defects or fetal death comes from the fact that VDTs emit ionizing radiation, which has been implicated in birth defects and increased fetal death rates. None of the numerous studies on emissions from VDTs (249) report levels of ionizing radiation that are known to be associated with biological effects of any kind. Since the primary emissions from VDTs are below 300 kHz, there is a possibility that low-level nonionizing radiofrequency radiation may be involved in some type of as-yet-unexplained adverse effect on reproduction. Great care should be taken in drawing any such inference, however, since no clear evidence exists to support any such association.

Information regarding the effects of low frequency electromagnetic radiation on reproduction in females is conflicting. Early studies with mice exposed continuously to 60 Hz electric fields (3.5 kV/m, 10 kV/m, and 15 kV/m) over several generations indicated that mortality in the offspring may be higher in certain exposed groups

Photo credit: Pemina Meisels

Reports of reproduction system effects among users of the many video display terminals (VDTs) now in use in the Nation's workplaces have raised questions about the safety of prolonged VDT exposure. Comprehensive studies of these effects are now in progress.

(223). A 1980 study reported no effects on fertility or development of offspring in mice exposed to a 240 kV/m 60 Hz electric field for about 3 months (105). Similarly, in rats exposed for 30 days to a 100 kV/m, 60 Hz electric field, no effect was seen on reproductive performance of the exposed animals, nor were significant adverse effects noted in the offspring (33).

NIOSH has undertaken an extensive study that is designed to help resolve the question of whether VDT use affects reproduction. The 3-year study will involve a cohort of 2,000 VDT-exposed women and 2,000 nonexposed controls. All women will be employed in nonmanagement positions in a small geographic area. Reproductive, health, and work histories will be obtained by self-administered questionnaires completed at three 9-month intervals. Personal habits such as alcohol, tobacco, and caffeine use will be taken into account. NIOSH intends to perform a follow-up study to evaluate future reproductive outcomes. Specific studies of adverse reproductive effects in men exposed to VDT emissions have not been conducted by NIOSH, nor are any being planned.

Another prospective study of 10,000 office workers has been initiated by Mount Sinai School of Medicine in cooperation with the Service Employees International Union and the 9 to 5 Association of Working Women. The study will be comprised of male and female VDT worker volunteers who will be compared with a group of non-VDT workers. Participants will complete extensive health quesionnaires on a regular basis. Results will be analyzed after 2 years. Follow-up studies are planned to determine whether children of VDT workers suffer an increased incidence of cancer (272). (A discussion of reproductive and other health effects of VDT emissions appears in OTA's upcoming report, *Automation and America's Offices*.)

Magnetic Fields

Magnetic fields are associated with power transmission lines, electric machinery and appliances, and the Earth's natural electric field. Beyond the near field region, an electric field is always associated with a complementary magnetic field, and vice versa.

The magnetic field strength directly beneath a 60 Hz alternating current (AC) power transmission line ranges from 0.3 to 0.6 G, dropping off to about 0.01 to 0.1 G 200 feet from the right-of-way center (82). By comparison, the Earth's natural magnetic field strength is 0.6 G, and localized 60 Hz magnetic fields around household appliances (e.g., color television sets, hair dryers) may range from 1 to 25 G. It is generally assumed that the biological effects of magnetic field are attributable to induced body voltage, electric fields, and currents.

Considerable interest has developed in recent years in evaluating the biological activity of low-level, low-frequency (50 to 60 Hz) magnetic fields. Exposure to this type of electromagnetic radiation commonly occurs in the vicinity of extremely low-frequency (ELF) communications antennas, which would result in significant population exposures.

Teratogenic effects in humans have not been associated with exposure to magnetic fields. Several studies in the United States, Sweden, England, and Wales have reported correlations between increased incidence of leukemia and possible exposure to electric and magnetic fields near high-voltage power lines. However, this association was not substantiated by a 1980 study (115). Information regarding effects of low-frequency electromagnetic radiation on reproduction in female laboratory animals is conflicting (105,223,330). Although these data do not permit a firm conclusion, they suggest that occupational exposures to magnetic fields may not constitute a hazard to reproduction.

Hyperbaric and Hypobaric Environments

Air pressures in excess of those found at sea level (14.7 pounds/square inch) are considered hyperbaric, and air pressures below that found at sea level are hypobaric. Workers exposed to hyperbaric environments include those engaged in caisson or tunneling operations, where compressed gas is used to exclude water or mud and to provide structural support during construction. Such operations are associated with pressures that can be more than four times that occurring at sea level (363). Underwater diving can be associated with considerable pressure, since each 10-meter increase in sea-water depth is equivalent to an increase of one atmosphere pressure. The primary health effect caused by hyperbaric environments is the tissue damage that results from expansion or contraction of gas spaces found within or adjacent to the body, such as around the teeth, in the sinuses, and within the ear. This type of effect is referred to as barotrauma. Other secondary types of damage caused by hyperbaric environments result from the narcotic action of nitrogen at four atmospheres of pressure or more, oxygen poisoning when its partial pressure exceeds two atmospheres, and the severe effects of rapid decompression.

Hypobaric environments can be of two types, high-altitude and low-altitude. High altitude hypobaric environments occur when pilots and air crews operate aircraft at altitudes in excess of 30,000 feet. In these situations, the greatest hazard is caused by lack of oxygen (hypoxia). Hypoxia also occurs at lower altitudes, as shown by the syndrome of impaired judgment and performance and general feeling of malaise associated with acute mountain sickness (363).

Male.—Only limited data are available on the influence of atmospheric pressure on male reproductive function. One study has described the semen characteristics of nine men exposed to high altitude (14,000 feet) in Peru for 4 weeks (83). A continuous decrease in sperm count was observed throughout the experiment. In addition, increased numbers of sperm abnormalities, decreased motility, and decreased testosterone levels were associated with high altitude. The principal causative factor for these changes may have been reduced ambient oxygen levels.

From the limited data available, it appears that male fertility can be suppressed by both hypobaric and hyperbaric environments (83). A 1968 review cited studies in which brief exposures to high altitude were found to cause impaired spermatogenesis, destruction of germinal epithelium, and testicular atrophy in several species of animals. These changes are apparently reversible on descent to sea level.

In a 1982 study, mice were exposed to high pressure (50 ATA) at intervals throughout one spermatogenic cycle and then mated with untreated females in order to evaluate effects on sex drive and fertility (20). A significant effect on male fertility resulted, as evidenced by reduced pregnancy rates in mated females. In addition, there was a reduction in live litter size, although no indication of teratogenic effects was obtained. The precise mechanism for the action of high pressure on male fertility could not be identified, especially in view of the fact that no gross morphological abnormalities were seen in the sperm.

Female.—Data concerning the effects of hyperbaric environments on female reproduction are limited to two case reports (40,357); there is thus insufficient scientific evidence to determine whether hyperbaric environments represent a hazard to female reproduction.

Pregnancy.—Atmospheric conditions are known to affect the outcome of pregnancy. Several studies have documented that human birth rates and birth weights are reduced in communities at high altitudes (75).

There is little information available regarding the effects of atmospheric pressure variations on female reproduction. Several studies have been conducted in pregnant dogs and sheep using conditions designed to simulate underwater diving and rapid decompression (258,340). In general, it appears that in the late stages of fetal development, the fetus appears to be less susceptible to decompression sickness than the mother. These studies do not provide an indication of the possible effects of hyperbaric exposures on the embryo/fetus early in pregnancy.

Hot and Cold Environments

The relationship of body heat to the external environment is a function of air temperature, air velocity, moisture content of the air, and radiant temperature. The hazards of working in a hot environment result when an imbalance occurs between metabolic heat production and heat loss from the body to the environment; i.e., heat loss fails to keep pace with heat produced by the body. A rise in body temperature is an indication that the body is storing heat that it cannot dissipate. As a result of the body's inability to adequately dissipate excess heat, four primary illnesses may occur. In order of increasing severity they are referred to as heat rash, heat cramps, heat exhaustion, and heat stroke. Heat stroke is a serious medical condition that can be fatal if not treated immediately. It is recommended (364) that workers should not continue to perform tasks that cause their body temperatures to exceed 38° C.

Maintenance of heat balance in a cold environment requires that the body restrict heat loss and increase heat production. The primary mechanism for limiting heat loss is constriction of the blood vessels (vasoconstriction), particularly in the extremities. This results in a drop in skin temperature and consequently less heat loss to the environment. Under severe conditions, the chilling of the extremities is so great that tissue freezing occurs, which results in frostbite. Work in a cold environment of sufficient duration to result in exhaustion will make the individual more prone to heat loss and the development of severe acute effects of general body hypothermia (364).

Experts have questioned whether women are exposed to work environments that are sufficiently hot to affect reproduction (75). Animal studies indicate that maternal temperature must be raised to at least 38.9° C before effects on the fetus are observed. Teratogenic effects have occurred in humans in conjunction with maternal hyperthermia. Prolonged fever in the mother during the first trimester of pregnancy appears to be a major factor in producing severe central nervous system dysfunction in offspring (70,110, 286). There is no documentation available concerning the specific effects on reproductive function or pregnancy outcome in women exposed to cold environments.

Although hyperthermia is well-known for its antispermatogenic effects in humans, there are no data available on the influence of cold environments on reproductive function in men. Documentation on the suppression of spermatogenesis by heat is largely related to certain medical disorders, such as cryptorchidism (undescended testes) varicocele (enlarged veins in the scrotum), and acute febrile illness (301). There are no case reports or epidemiologic studies of reproductive function in men working in hot environments. One group of experts has concluded that the occurrence of adverse reproductive effects in men from exposure to hot environments is unlikely under normal working conditions (75). Occupational exposure to direct heat, by contrast, may be a leading cause of male infertility.

Application of heat to the scrotum has been promoted as an effective, reversible means of male birth control. In controlled studies with human volunteers, elevation of testicular temperature by 2.5° to 3.0° C for 30 minutes on several alternate days led to depression in sperm count beginning at 3 weeks after exposure and lasting 3 to 5 weeks (299,301). Sperm counts subsequently recovered and in fact increased beyond pre-exposure levels. It is important to note that these transient decrements in sperm count are unlikely to be associated with a decrease in male fertility and should not be used as a contraceptive method.

Noise and Vibration

Noise, generally identified as unwanted sound, is probably the most prevalent of all occupational hazards. Permanent, noise-induced hearing loss has been recognized for several hundred years (5). Noise is classified according to several criteria. Wide-band noise refers to sound that covers a large portion of the available frequency spectrums, and is typified by the noise produced by large machinery and jet engines. Narrow-band noises are often associated with a definite pitch, such as that produced by a circular saw or other power-cutting tools. A noise of short duration (less than a second) that rises rapidly to a peak and then falls to below background levels is referred to as impulsive or impact noise. The sounds of a gunshot or a forging hammer are examples of impulsive noise.

Vibration occurs in all segments of industry in which power-driven tools, heavy machinery, and mechanized equipment are utilized. When considering workplace exposure, vibration is usually categorized as either segmental or whole-body vibration. Whole-body vibration is mechanically transmitted to the entire human body through a supporting structure, such as a vehicle seat. Segmental vibration affects localized parts of the body, usually the hands and feet. Hand-operated tools are a common source of segmental vibration.

The harmful effects of segmental vibration appear to be more severe than for whole-body vibration (364). Workers who use vibratory hand tools for prolonged periods may develop Raynaud's phenomenon ("dead hand" or "vibratory white fingers"). This condition is associated with numbness and blanching of the fingers, and can result in loss of muscular control and reduced sensitivity to vibration, pain, and temperature. Numerous additional ailments can be associated with segmental vibration, including changes in bone, nerve degeneration, muscular weakness and atrophy, and Dupuytren's disease, which causes permanent flexion of one or more fingers (364).

Male.—There is no evidence to indicate that occupational exposure to noise is harmful to male reproductive function, nor is there conclusive evidence of adverse effects of vibration on reproductive function in men. One report found sperm abnormalities and decreased fertility among professional drivers, which may have resulted from vibration (75). Other factors, however, including elevated intrascrotal temperature from prolonged sitting, may also be implicated.

Female.—Evidence concerning the effects of noise on reproductive function and pregnancy outcome in humans is largely circumstantial and conflicting. No information is available on the effects of occupational exposure to noise. Based on the results of animal studies, it is presumed that vibration may affect the human embryo. There are no specific reports of adverse reproductive effects in the human female resulting from vibration.

Pregnancy.—The most consistently reported reproductive effect of noise in animals is pregnancy-rate reduction (253). In addition, there is evidence that embryolethality and fetolethality are increased by noise exposure. Both positive and negative findings with respect to teratogenesis have been reported. Differences in noise level and variations in spectral and temporal patterns of exposure may all be expected to influence the biologic effect produced. Thus far, it has not been possible to use the results from available animal studies to predict whether similar effects may occur in humans.

There are insufficient data available from animal studies to critically evaluate the reproductive effects of mechanical vibration. In a 1971 study, pregnant mice were exposed at 4¼ and 7 days gestation to whole-body vibration for 10 minutes at 3 different frequencies (5 Hz, 10 Hz, 20 Hz) (24). Mouse embryos were found to be quite resistant to vibration, although in the 4¼ day embryos, the incidence of abnormalities was increased in the 20 Hz group.

EFFECTS OF STRESS ON REPRODUCTIVE FUNCTION

Stress, in the workplace as elsewhere, refers to a type of individual response to an environmental stimulus or condition. The principal sources of stress in the work environment are posture, work on industrial machines, physical exertion, mental stress, environmental factors, and characteristics of the worker (174,220).

Psychological Stress

It has long been suspected that psychological stress may lead to infertility in both men and women. This possibility seems clear from the evidence in animals. The question of a relationship between psychological variables and infertility is complex, and the literature, although extensive, is speculative, anecdotal, and contradictory. Few studies meet adequate methodological standards. Nevertheless, a consideration of the effects of workplace stress on reproductive function must address the question of psychogenic infertility.

Psychological stress can lower testosterone levels (191) and may be associated with decreased sperm counts (35,229). In women, stressful experiences, such as those encountered in wartime, may lead to amenorrhea (182). Clinical evidence of such stress-induced psychological endocrine reactions among patients attending infertility clinics is anecdotal. Hence, although a psychological mechanism associated with infertility is possible, there is little firm evidence of stress-induced infertility, save for some cases of amenorrhea. Knowledge of psychogenic endocrine reactions is extremely limited (33).

Workplace psychological stress may play a role in infertility by means of a behavioral mechanism—through interference with the sexual relationship. In this context, the following sexual problems have been cited: impotence, retarded ejaculation, ejaculation prior to intromission, infrequent intercourse, and vaginismus (extreme aversion to coitus accompanied by painful spasm of the vagina) (94). Detection of behavioral problems induced by psychological stress depends on a number of factors, including the comfort of the clinician in asking, and the comfort of the patient in answering, detailed questions about sexual behavior (54), and how extensive an assessment of sexual function is made (33).

Further study may reveal that the reproductive status of workers facing workplace-induced psychological stress exhibits a distribution that mirrors that of the population at large. Adaptation to psychological stress may not represent the demands of a particular stress, such as job insecurity or long working hours, but rather the manifestations of enduring personality constructs and capabilities (60).

Physiological Response to Stress

Stress, from whatever source, stimulates several hormonal responses in both women and men. Prominent among these responses are the secretion of ACTH (adrenocorticotropin, a hormone stimulating the adrenal glands) from the pituitary gland, and neurotransmitters and steroid hormones from the adrenal glands. These hormones serve to adapt the body to stress ranging from the mildly psychological to the intensely physical by affecting the cardiovascular, energy-producing, and immune systems (17).

Plasma levels of the neurotransmitters epinephrine and norepinephrine are one measure of stress-induced activation of the adrenal glands and nervous system. Until recently, it was difficult to obtain a reliable measure of plasma epinephrine and norepinephrine because of their extremely low concentrations in the blood. The introduction of highly sensitive assays, however, has made it possible to determine their concentrations during stressful situations in humans.

Physical exertion, cold, and heat stress, for example, can cause marked elevations in these hormones. Public speaking may result in a 50 percent increase in plasma norepinephrine and a 100 percent increase in plasma epinephrine (17).

Proper reproductive function is also heavily dependent on the functional integrity of these three major body systems. As the complex hormonal and biochemical sequelae of workplace stress become better known, it is likely that a more complete understanding of the effects of workplace stress on reproductive function will emerge. At present, the documented effects of repeated or prolonged stress on the cardiovascular, energy-producing, and immune systems should be regarded as factors with the potential to compromise reproductive function.

Physical and Psychological Stress and the Pregnant Worker

The pregnant employee is able, in most cases, to continue productive work until the onset of labor at 40 weeks (168). It is important to note that in a discussion of pregnancy and working, generalizations are made only for normal, uncomplicated pregnancies. Complications of pregnancy (e.g., vaginal bleeding, premature rupture of the membranes) (29) may cause some women to modify certain aspects of their work at specific times during their pregnancies.

Recent research offers reassurance that working during pregnancy is not in itself a risk factor for adverse outcome. Pregnancy outcomes of 7,155 women who worked between 1 and 9 months of pregnancy were compared with outcomes of 4,018 women who were not employed during pregnancy (222). It is significant that no differences were found between the group of working pregnant women and the group of nonworking pregnant women in rates of premature birth, Apgar score, perinatal death rate, birth weight, use of special care nurseries, or prevalence of malformations. These findings indicate that working to term in the absence of contraindications does not impose an added risk on mother or infant. Remaining unanswered is the question of whether any specific occupational groups are at increased risk of adverse pregnancy outcome by virtue of their continued employment during pregnancy.

In a 1982 study comparing pregnant and nonpregnant women and their partners, pregnant women were more likely to report altered states; e.g., "feeling ill" or "feeling overweight." Pregnant women, however, reported the fewest impacts of these states on their performance in the workplace as compared with any of the other groups (prospective fathers, and nonexpecting women and men) (214).

Quantifying the relative risks posed by occupational stresses during pregnancy is particularly difficult because of the absence of baseline data for comparison. There has been no scientific study, for example, comparing the pregnant worker's exertion (mental or physical) during paid employment with that of full-time work in the home. Thus the relative risk to the pregnant worker from workplace stress versus stress in a nonoccupational setting cannot be readily evaluated. A job may entail strenuous activity, such as lifting, which the anatomical changes of pregnancy may make difficult to perform, although women who are accustomed to activities that may be strenuous to others may be able to continue their usual jobs virtually throughout their pregnancies.

The American Medical Association (7) has published guidelines for various job tasks during pregnancy. Table 4-4 shows the period of time during which healthy employees with normal, uncomplicated pregnancies should be able to perform specific tasks without undue difficulty or risk to the pregnancy. All pregnant employees need not stop these activities at the exact time of gestation noted, but the guidelines may be used to help evaluate individual cases. In addressing the issue of the pregnant worker, the American College of Obstetricians and Gynecologists makes the following recommendation:

> The normal woman with an uncomplicated pregnancy and a normal fetus in a job that presents no greater potential hazards than those encountered in normal daily life in the community may continue to work without interruption until the onset of labor and may resume working several weeks after an uncomplicated delivery (6).

Table 4-4.—Guidelines for Continuation of Various Job Tasks During Pregnancy

Job task	Week of gestation
Secretarial and light clerical	40
Professional and managerial	40
Sitting with light tasks:	
Prolonged (more than 4 hours)	40
Intermittent	40
Standing:	
Prolonged (more than 4 hours)	24
Intermittent:	
More than 30 minutes per hour	32
Less than 30 minutes per hour	40
Stooping and bending below knee level:	
Repetitive (more than 10 times per hour)	20
Intermittent:	
2 to 10 times per hour	28
Less than 2 times per hour	40
Climbing:	
Vertical ladders and poles:	
Repetitive (4 or more times per 8-hour shift)	20
Intermittent (less than 4 times per 8-hour shift)	28
Stairs:	
Repetitive (4 or more times per 8-hour shift)	28
Intermittent (less than 4 times per 8-hour shift)	40
Lifting:	
Repetitive:	
Less than 25 lb.	40
25 to 50 lb	24
More than 50 lb	20
Intermittent:	
Less than 25 lb	40
25 to 50 lb	40
More than 50 lb	30

SOURCE: American Medical Association Council on Scientific Affairs, "Effects of Pregnancy on Work Performance," *J.A.M.A.* (251):1995-1997, 1984.

EFFECTS OF WORKPLACE BIOLOGICAL AGENTS ON REPRODUCTIVE FUNCTION

Occupations associated with a risk of an infectious disease fall into two categories: 1) health care occupations, with direct patient contact, laboratory exposure to infective material, or production of biological materials, and 2) nonhealth care occupations, primarily those involving contact with animals or animal products, refuse collection, groundbreaking or earthmoving, individuals in nonmedical settings (e.g., social workers), or travel into areas of endemic disease. Most of the available information about workplace biological hazards to reproductive function concerns workers in the first category.

Among health care workers, the hazards of hospital-acquired, or nosocomial, infectious diseases have long been recognized. Less attention has been given to such problems among those in outpatient settings; e.g., dentists' and doctors' offices, kidney dialysis centers, laboratories where there is contact with blood, nursing homes, institutions for the retarded, and prisons (118).

Health care personnel are frequently exposed to infectious agents that can cause intrauterine infections, produce teratogenic effects in their offspring, be passed to and infect their offspring, or act as abortifacients. These agents include the viruses rubella, cytomegalovirus, and hepatitis B. Some infectious agents may also infect and impair male reproductive function (e.g., mumps, orchitis).

Rubella

Rubella, or German measles, is a virus that threatens health care workers and certain nonhealth care workers, such as school teachers and day-care workers, who are likely to have contact with children infected with the disease. The major hazard of rubella is infection in pregnant women, with the possibility of congenital rubella syndrome developing in their offspring. Transplacental infection of the fetus in the first trimester produces developmental abnormalities of the heart, eyes, brain, bone, and ears, often without interrupting the pregnancy. Congenital rubella is also associated with developmental abnormalities of the male reproductive system (291). Intrauterine infection may also result in miscarriages and stillbirths.

The widespread use of rubella vaccine has greatly reduced the incidence of the disease in the United States. In 1984, 959 cases of rubella and 4 cases of congenital rubella syndrome were reported in the United States (243). Thirteen States and the District of Columbia reported no rubella cases, and 284 of 3,137 U.S. counties (91 percent) were free of rubella in 1983 (241). Whether occupational exposure to the production or formulation of rubella vaccine has produced congenital infections is not known.

Depending on the severity of the illness, the costs of caring for an infant with congenital rubella can be substantial. Such costs can include hospitalization for treatment and repair of congenital heart lesions and cataracts, special educational services, and institutionalization for the most severely affected children (275). The existence of even a limited number of cases of congenital rubella syndrome is thus of significant economic consequence. The average lifetime cost for a child with congenital rubella syndrome is estimated to be $221,660 in 1982 dollars (185).

Therapeutic abortion may be a consequence of rubella infection of pregnant women. Limited information suggests that rubella-associated abortions are considerably more common than cases of congenital rubella syndrome. In an outbreak of rubella in Hawaii in 1977, 11 of 12 women who had rubella elected to undergo abortion (224,322).

Rubella vaccine is the most effective means of preventing the disease. It is well tolerated in the work setting and results in minimal absenteeism (118). The authors of a 1984 study (275) declare that the opportunity is at hand to eliminate rubella from the United States by ensuring that susceptible females of childbearing age are vaccinated and by requiring proof of rubella immunity for all children enrolled in schools.

Cytomegalovirus

Cytomegalovirus, a member of the family of herpes viruses, is generally of minor consequence in normal populations, and infection may be asymptomatic. It can have a major impact, however, if contracted during pregnancy. For this reason, the risk of acquiring cytomegalovirus is of serious concern to many female health care workers. Intrauterine infection with transmission to the fetus is one of the most serious consequences of cytomegalovirus infection in women. Offspring of infected mothers may have an enlarged liver, an enlarged spleen, microcephaly (abnormally small head), microphthalmia (abnormally small eyes), and mental or motor retardation.

Infants who are infected with cytomegalovirus shed large quantities of virus into their urine and saliva. Because these infants commonly have no symptoms attributable to cytomegalovirus, the viral infection is likely to go undetected. Nursery and pediatric health care personnel and teachers in day-care centers are frequently exposed to the secretions of infected newborns and older infants. Yet evidence indicates that this occupational contact confers no greater risk than that faced by young women in the community at large. Thus, although female health care workers frequently and unknowingly care for infants shedding cytomegalovirus, and exhibit a high degree of concern about this exposure, their incidence of primary infection is not higher than that of other young women (87). Data from experimental animals suggest that the ovary or testis may serve as a reservoir for cytomegalovirus (43,86).

Hepatitis B

Hepatitis B is the most dangerous form of hepatitis, a debilitating liver disease characterized by fever, weakness, loss of appetite, headache, and muscle pain. There are nearly 1 million hepatitis B virus carriers in the United States today, and the cost of hepatitis B infection in this country is estimated to be $1 million per day. Up to 1 percent of those infected with hepatitis B may die of the disease, and 5 to 10 percent of infected persons become chronic carriers of the virus who can remain infectious indefinitely (128). Once infection with hepatitis B occurs, there is no known treatment.

Contact with infected blood or saliva is the essential factor in occupational acquisition of hepatitis B virus. The groups at highest risk for acquiring hepatitis B virus are medical technicians, operating room staff, phlebotomists, physicians (especially surgeons and pathologists), nurses (particularly intravenous-therapy nurses, and nurses in oncology and dialysis units), dentists and oral surgeons, laboratory and blood-bank technicians, and emergency-room staff. Morticians and their assistants who have routine contact with blood and secretions are also at high risk of hepatitis B infection (242).

Workers may acquire hepatitis B virus via accidental needle punctures, touching the mucous membranes of the nose, rubbing the eyes, and from human bites that penetrate the skin (238). Those routes serve to infect workers of both sexes; there are added consequences if pregnancy ensues.

Transmission from mother to infant during or following birth is an efficient mode of hepatitis B virus transmission; between 10 and 50 percent of infants born to mothers infected with the disease may also become infected (62,308). The risk of postnatal infections can be diminished with the use of hepatitis B immunoglobulin (80,248,308). Although infection is rarely symptomatic in the acute phase, approximately 90 percent of infected infants will become chronic hepatitis B carriers. This presents a double-barreled public health problem: 1) female carriers may subsequently perpetuate the cycle of perinatal transmission, and 2) chronic hepatitis B infection is associated with hepatocellular carcinoma, a form of liver cancer (136).

Other Infectious Agents

Several other infectious agents to which health care personnel may be exposed in the workplace, either in the form of infected patients or contaminated body fluids, may have untoward consequences for pregnant workers, or workers who later become pregnant (389). The principal infectious agents in this group are:

- **Herpes simplex virus,** which may produce microcephaly (abnormally small head), microphthalmia (abnormally small eyes), and retinal defects in the offspring of infected women. Typical herpes lesions have been noted in newborns of infected mothers, and the virus has been isolated from the placenta. These effects are due to exposure of the neonate to active genital lesions at the time of delivery. Herpes simplex viral infection has recently come under suspicion as a cause of previously unexplained spontaneous abortions (129).
- **Congenital syphilis,** a bacterial infection, which causes numerous abnormalities in the skin, mucous membranes, skeleton, nervous system, and eyes in infants born to infected women.
- **Toxoplasmosis,** caused by a protozoan organism, which can cause macro- or microcephaly, microphthalmia, and mental deficiency in babies born to infected mothers.
- **Varicella,** or chicken pox, caused by the herpes varicella-zoster virus, which can produce skin scars, limb deformities, microphthalmia, cataracts, and mental deficiency in infants exposed in utero during pregnancy.

It is important to note that problem pregnancies caused by infectious agents are relatively rare. The overwhelming majority of women with herpes simplex or herpes zoster infection during pregnancy, for example, give birth to normal babies.

Recombinant DNA

The rapid expansion of the field of biotechnology (360) will increase potential exposures of skilled and unskilled workers to: 1) micro-organisms containing recombinant deoxyribonucleic acid (DNA), and 2) their products. Micro-organisms have for centuries been employed for leavening bread, fermenting beer and wine, and ripening cheese. These traditional applications depend on naturally occurring mutations to provide microbial strains with particularly useful properties. Modern biotechnology, however, takes advantage of recent advances in molecular genetics and cell biology to expand the use of micro-organisms. Genetic manipulation of molecules of DNA to form new, recombinant DNA permits the development of novel micro-organisms (196).

Under present working conditions, the threat, if any, to reproductive function of occupational exposure to genetically altered micro-organisms appears to be slight. Many micro-organisms currently used in biotechnology are "attenuated," or debilitated, through genetic manipulation, so that their ability to reproduce outside of carefully controlled culture conditions is severely curtailed. None of the organisms in use today have been shown to cause either infection or disease in workers using the techniques of biotechnology (196).

Any reproductive hazards of occupational exposure to the biologically active products of recombinant micro-organisms are not a consequence of recombinant DNA techniques per se. Product hazards in biotechnology are not likely to differ qualitatively from those encountered in other sectors of the pharmaceutical and chemical industries. The fact that the molecules encountered in biotechnology are the products of engineered micro-organisms, rather than naturally occurring ones, or of synthetic catalysis, will not alter their reactivity or toxicity. For example, the synthetic manufacturing and packaging of estrogenic hormones has produced excessive breast development, or gynecomastia, in male workers (140). Use of engineered micro-organisms to manufacture these hormones is likely to result in a hazard of similar nature. Exposure to biologically active products constitutes a class of potential hazards throughout the chemical and pharmaceutical industries, and biotechnology applications are not likely to be exempt from such hazards (196).

To the extent that biotechnology uses highly specific techniques to produce particular chemicals, it will decrease the number of currently encountered mixtures of chemicals—sometimes contaminated with toxic compounds—that are common in conventional chemical synthesis. It is noteworthy, too, that recombinant DNA technologies, operating at moderate temperature and pressure, have fewer inherent physical hazards than traditional chemical syntheses (360).

SUMMARY AND CONCLUSIONS

Two elements are required to constitute a workplace hazard to reproductive health. First, a worker (and perhaps a developing embryo/fetus) must be exposed to a chemical, physical, or biological agent. Second, the agent must be toxic to reproductive function or embryonic/fetal development.

Identifying exposed workers, evaluating their level of exposure, and determining their degree of reproductive impairment—if any—continues to be difficult. Studies of experimental animals offer valuable indicators of potential workplace reproductive hazards, but the extrapolability of animal studies to humans is variable.

Although present knowledge is incomplete, concern about workplace chemical hazards to reproductive function has focused on metals (lead, mercury, cadmium, arsenic, antimony, boron, and manganese), agricultural chemicals (carbaryl, DBCP, DDT, chlordecone, 2,4,5-T, dioxin, 2,4-D, PBB, and PCB), organic solvents, anesthetic agents, epichlorohydrin, EDB, EtO, formaldehyde, rubber (1,3-butadiene, chloroprene, and ethylene thiourea), vinyl halides, hormones, and other undefined industrial exposures. Review of these compounds reveal that there is indeed cause for concern about reproductive hazards resulting from occupational exposures.

Present knowledge is also incomplete for physical factors of potential concern, including non-ionizing electromagnetic radiation, atmospheric or ambient pressure (hypobaric and hyperbaric environments), heat, cold, noise, vibration, and stress. Although there is extensive evidence available for the harmful effects of ionizing radiation, the effects of occupational exposure have not been well researched.

Workplace stress refers to: 1) an environmental condition, 2) a worker's response to that condition, or 3) a relationship between the environmental demands and a worker's ability to meet those demands. The elements of occupational stress are posture, work on industrial machines, physical exertion, mental stress, environmental factors, and characteristics of the worker. Aside from imposing physical stressors, workplace activities may lead to psychological stress. Both physical and psychological stress are thought to be sources of worker infertility, although direct evidence of this phenomenon has proven elusive.

Biological agents—agents of infectious disease—are a potential reproductive hazard to those in health care occupations, either through direct patient contact, through laboratory exposure to infective material, or through exposure to materials on infected individuals. Exposure to the viruses rubella, cytomegalovirus, and hepatitis B is of concern, as is exposure to such infectious agents as herpes simplex virus, congenital syphilis, toxoplasmosis, and varicella through contact with either infected patients or contaminated body fluids.

Most available data only suggest that certain occupations or occupational exposures are associated with adverse effects on male or female reproduction, or fetal development. In some cases it is possible to identify the site and mechanism of reproductive toxicity. In most instances, however, the gaps in information are enormous.

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LIST OF CHEMICAL NAMES

Lead
Also known as, or contained in: lead azides, lead salts, lead tetraethyl, lead tetramethyl, metallic lead, TEL, tetraethylplumbane, TML, and tetramethylplumbane.

Boron
Also known as boric acid, orthoboric acid.

Manganese
Compounds include manganese acetate, borate, bromide, carbonate, carbonyl, chloride, difluoride, dioxide, hypophosphite, iodide, nitrate, oleate, oxalate, oxide, phosphate (dibasic), pyrophosphate, selenide, sesquioxide, silicate, sulphate, sulphide, trifluoride.

Mercury
Also known as hydrargyrum, liquid silver, quicksilver; compounds include mercuric acetate, arsenate, bromide, chloride, chloride (ammoniated), cyanide, dichromate, fluoride, iodate, iodide, nitrate, oxide (red), oxycycanide, subsulphate, sulpate, sulpide, (red), thiocyanate; mercurous acetate, bromide, chlorate, chloride, fluoride, iodide, nitrate, sulphate.

Cadmium
Compounds include cadmium acetate, carbonate, chloride, fluoroborate, fluoride, molybdate, nitrate, oxide, sulphate, sulphide.

Arsenic
Also known as arsen, arsenic black, gray arsenic, metallic arsenic; compounds include arsanilic acid; arsenic pentoxide, sulphide, trioxide; arsine; calcium arsenate; dimethylarsinic acid; lead arsenate; methanearsonic acid (disodium and monosodium salt); potassium arsenate; potassium arsenite; sodium arsenate, arsenite, cacodylate.

Carbaryl
Also knonw as, or contained in 1-naphthyl-N-methyl carbamate, 1-naphthyl methyl carbamate, nitrosocarbaryl, and Sevin.

Dibromochloropropane
Also known as, or contained in 1,2-dibromo-3-chloropropane, 3-chloro-1,2-dibromopropane, Fumazone, Nemazon, and Nemaset.

Kepone (Chlordecone)
Also known as, or contained in Acarin, Kelthane, and Mitigan.

Polybrominated Biphenyls (PBB)
Also known as, or contained in decabromobiphenyl, decabromodiphenyl, hexabromobiphenyl, hexabromodiphenyl, octobromobiphenyl, octabromodiphenyl, and perbromobiphenyl.

Polychlorinated Biphenyls (PCB)
Also known as or contained in askarels, Aroclor, Chlophen, Chlorextol, chlorinated biphenyl, chlorinated diphenyl, chloro-biphenyl, Dykanol, Fenclor, Inerteen, Kanechlo, Noflamol, Phenoclor, polychlorinated biphenyl, polychlorobiphenyl, Pyralene, Pyranol, and Santotherm.

Epichlorohydrin
Also known as, or contained in 1-chloro-2,3-epoxypropane, 3-chloro-1,2-epoxypropane, 3-chloro-1,2-propylene oxide, (chloromethyl) ethylene oxide, (chloromethyl) oxirane, 2-chloromethyl oxyrane, 3-chloropropene-1,2-oxide, chloropropylene oxide, g-chloropropylene oxide, ECH, ECHH, a-epichlorohydrin, 1,2-epoxy-3-chloropropane, 2,3-epoxypropyl chloride, glycerol epichlorohydrin, glycidyl chloride, and SKEKhG.

Ethylene Dibromide
Also known as, or contained in Aadibroom, Bromofume, Celmide, dibromoethane, 1,2-dibromoethane, sym-dibromoehtane, Dowfume EDB, Dowfume MC-2, Dowfune W-8, Dowfune W-85, Dowfune 40, E-D-BEE, EDB-85, ENT 15, 349, ethylene bromide, Fumo Gas, glycol dibromide, Iscobrome D, Kopfume, Nefis, Pestmaster, Pestmaster EDB-85, Sanhyuum, Soilbrum-40, Soilbrum-85, Soilfume, and Unifume.

Ethylene oxide
Also known as, or contained in Anprolene, Benvicide, Carboxide, Cry-oxide, dihydrooxirene, dimethylene oxide, epoxyethane, 1-2-epoxyethane; EO, ETO, oxacyclopropane, Oxane, oxidoethane, a,B-oxidoethane, Oxiran, Oxirane, Oxyfume, Oxyfume 12, Oxyfume sterilant-20, Pennoxide, Steroxide-12, Steroxide-20, and T-gas.

Formaldehyde
Also known as, or contained in BFV, Fannoform, Formalin, Formalith, formic aldehyde, Formol, Fyde, HCHO, Ivalon, Karsan, Lysoform, Methanal, methyl aldehyde, methylene oxide, Morbicid, oxomethane, oxymethylene, Paraform, and Superlysoform.

Vinyl Chloride
Also known as, or contained in chlorethene, chlorethylene, chloroethene, chloroethylene, ethylene monochloride, monochloroethene, monochloroethylene, Tridene, Trovidur, VC, vinyl C monomer, and VCM.

Carbon tetrachloride
Also known as tetrachloromethane, Carbona, carbon chloride, carbon tet, methane tetrachloride, perchloromethane, tetrachlorocarbon.

Styrene
Also known as ethenylbenzene, Cinnamene, phenethylene, phenylethene, phenylethylene, styrol, styrole, styrolene, vinylbenzene, vinylbenzol.

Xylene
Also known as dimethylbenzene, xylol.

Toluene
Also known as methylbenzene, toluol, methyl benzene.

Benzene
Also known as benzin, benzine, benzol, benzole, benzolene, bicarburet of hydrogen, carbon oil, coal naphtha, cyclohexatriene, motor benzol, phene, phenyl hydride, mineral hnaphtha, pyrobenzol, pyrobenzole.

Chapter 5

Technologies for Assessing Human Reproductive Function

CONTENTS

Page

Introduction 129

Tests of Male Reproductive Health 131
  The Fertility Evaluation 131
  Semen Quality 135
  Sperm Function 138

Tests of Female Reproductive Health 140
  Personal History 141
  Physical Examination 141
  Ovarian Function 141
  Cervical Mucus 145
  Endometrial Cells 145
  Tubal Patency/Uterine Structure 146
  Implantation/Establishment of Pregnancy 146
  Embryonic Differentiation and Fetal Development 146
  Delivery and Lactation 150

  Conclusion 151

Technical Notes 152

Chapter 5 References 155

List of Tables

Table No. *Page*
5-1. Patient History 132
5-2. Circumstances for Which Amniocentesis is Recommended 148
5-3. Use of Ultrasound in Fetal Monitoring 150

List of Figures

Figure No. *Page*
5-1. Chronology of Fertility Evaluation 130
5-2. Diagnostic Techniques in Fertility Assessment 133
5-3. A Gauge Used to Measure the Occurrence of Erection During Sleep 135
5-4. Sperm Agglutination 136
5-5. Sperm Movement Patterns 137
5-6. Sperm Morphology: Some Categories 138
5-7. The Morphology Overlay 139
5-8. The Menstrual Cycle 143
5-9. Basal Body Temperature Patterns Throughout the Menstrual Cycle 144
5-10. Cervical Mucus Ferning 145
5-11. Mean Human Chorionic Gonadotropin (hCG) During Pregnancy 147
5-12. Amniotic Cavity 147
5-13. Chorionic Villus Biopsy 149

Chapter 5

Technologies for Assessing Human Reproductive Function

INTRODUCTION

One of the clearest indicators of reproductive health in a population is the incidence of healthy offspring. Birth statistics can be misleading, however, in their failure to indicate the number of couples who are not engaging in procreation or are unable to reproduce. Although individuals who wish to have children often take for granted their physical ability to do so, exposure to certain chemical, physical, or biological agents can compromise reproductive health and sexual functioning (chapter 4 describes the effects of individual agents).

Because of the structural and functional differences between the sexes, exposure to one of these agents may impair the reproductive capacity of one sex and not the other. Individual characteristics and lifestyle differences (e.g., smoking, age, nutrition) also alter sensitivity to some agents. Monitoring the reproductive health of individuals exposed to known or suspected reproductive hazards is thus an important step.

The following section describes the diagnostic procedures available to patients experiencing reproductive health problems. **Although the emphasis is on infertility, it must be stressed that a thorough assessment of reproductive health includes factors not directly related to conception and fetal development (e.g., pubertal development, libido).** Reproductive health refers to the entire composite of human reproductive and sexual functions and their integration with other organ systems (see chapter 3).

It is critical to note that an individual's reproductive competence cannot be verified in isolation. Fertility is the product of the specific interaction of a couple. Physical examination and laboratory analyses may determine that a man or woman is *potentially* fertile (i.e., sound reproductive organs, normal hormone levels, presence of reproductive cells), but fertility is verified only after the couple has given birth to a healthy infant. Evaluation and treatment of infertility must therefore consider the couple as a unit. Ideal management is best achieved when the couple is seen together by a team of physicians (e.g., the man by a urologist, the woman by a gynecologist) (61).

Three features form the basis of a fertility evaluation in both men and women:

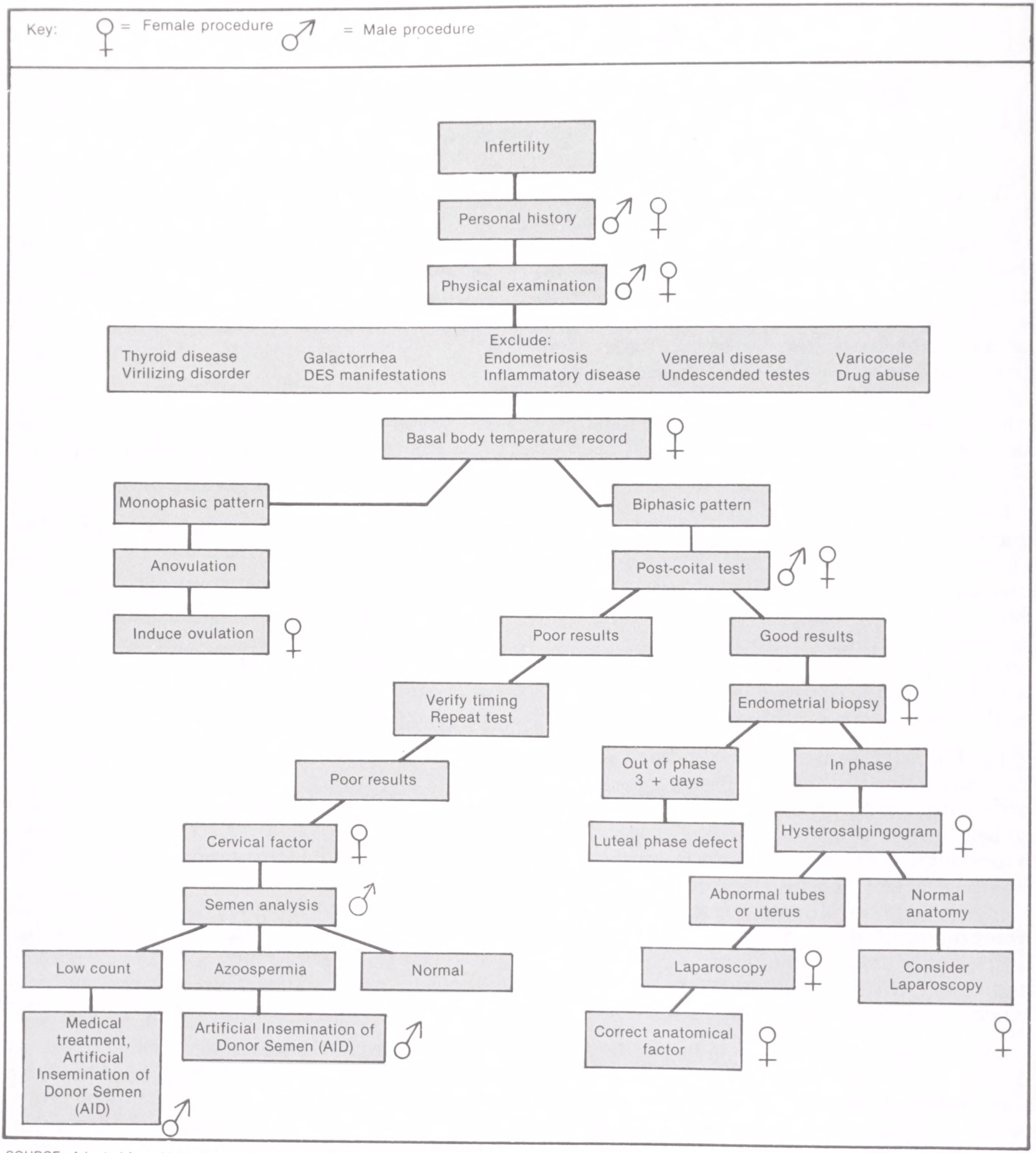
1. personal history (including medical, familial, occupational, and reproductive background);
2. physical examination; and
3. laboratory analyses (e.g., hormone studies, semen analysis, cervical mucus assays) (see figure 5-1).

Biological and practical considerations, however, demand that the parameters measured and the methods used be quite different for the two sexes. Whereas the male reproductive organs and germ cells (sperm) are readily accessible, the female correlates are not.

Physical examination of the male is simplified by the fact that his reproductive organs are external. Moreover, laboratory analysis of semen is a routine component of the male fertility evaluation. A man's ability to produce a semen sample and the analysis of various physical and functional properties of the sample provide an important indication of his reproductive health. These assays are safe, rapid, and easily performed with equipment standard to most hospitals and fertility clinics.

Unlike the male, the female reproductive organs are internal and her germ cells (eggs) do not leave her body. Direct observation of a woman's reproductive organs and germ cells, therefore, requires an invasive procedure or the use of special imaging equipment. The assessment of female reproductive competence thus relies heavily on indirect indicators. [An indirect indicator is defined here

Figure 5-1.—Chronology of Fertility Evaluation


Key: ♀ = Female procedure ♂ = Male procedure
Infertility
Personal history ♂ ♀
Physical examination ♂ ♀
Exclude:
Thyroid disease
Virilizing disorder
Galactorrhea
DES manifestations
Endometriosis
Inflammatory disease
Venereal disease
Undescended testes
Varicocele
Drug abuse
Basal body temperature record ♀
Monophasic pattern
Anovulation
Induce ovulation ♀
Biphasic pattern
Post-coital test ♂ ♀
Poor results
Verify timing
Repeat test
Poor results
Cervical factor ♀
Semen analysis ♂
Low count
Medical treatment, Artificial Insemination of Donor Semen (AID) ♂
Azoospermia
Artificial Insemination of Donor Semen (AID) ♂
Normal
Good results
Endometrial biopsy ♀
Out of phase 3 + days
Luteal phase defect
In phase
Hysterosalpingogram ♀
Abnormal tubes or uterus
Laparoscopy ♀
Correct anatomical factor ♀
Normal anatomy
Consider Laparoscopy ♀

SOURCE: Adapted from M. M. Seibel, "Infertility," in *Gynecologic Decision Making,* Emanuel A. Friedman (ed.) (Philadelphia: B. C. Decker Inc., 1983), pp. 68-69.

as one in which the reproductive endpoint under study is not observed, but its function is assumed based on the occurrence of related events. For example, while ovulation—the release of an egg cell from the female ovary—is not readily observable, there are associated changes in hormone levels and in body temperature that indicate when ovulation has occurred.] (A discussion of male and female reproductive function appears in chapter 3.)

Fertilization is but one of several events that are critical to successful reproduction. Others include transport of the fertilized egg to the uterus, implantation in the uterine wall, growth and development of the embryo/fetus, and delivery. Because each of the events subsequent to fertilization occurs within the female, the ability to accommodate and maintain a pregnancy is a component of female reproductive function.

As with other aspects of fertility, there is no absolute verification that can be made of a woman's ability to conceive and sustain a pregnancy, short of her actually doing so. However, several clinical techniques enable the physician to monitor these events as they occur. These may prove useful in isolating the effects of various agents on the reproductive health of exposed individuals or on their offspring. In-utero monitoring of embryo/fetal development, for example, may detect the effects of agents that do not impede conception, but that elicit structural or functional abnormalities in the offspring of those exposed.

The following discussion examines methods for assessing human reproductive health, including events preceding, following, and independent of fertilization. These are the diagnostic techniques used with patients experiencing reproductive health problems. While diagnosis of the physiological basis of a reproductive disorder does not necessarily identify its source (e.g., workplace exposure, lifestyle characteristic), tracing patterns in the incidence of reproductive problems (e.g., infertility, deformed offspring) may make these correlations possible. The use of epidemiology and animal toxicology studies to identify reproductive hazards is discussed in chapter 6.

TESTS OF MALE REPRODUCTIVE HEALTH

The Fertility Evaluation

Personal History

Obtaining a thorough personal history is the first and one of the most important steps in a fertility evaluation. Information about the individual's personal and familial health background and the couple's sexual interaction can provide important insights into the cause of infertility. Certain drugs, medical procedures, and diseases, for example, can compromise reproductive function. Coital method (e.g., use of certain vaginal lubricants, timing, position) can also contribute to fertility problems, as can certain personal practices (e.g., frequent exposure to excessive heat as from saunas and hot baths). It is also important for the physician to ascertain whether the patient has experienced any form of sexual dysfunction (e.g., impotence or decreased libido), whether the couple engages in intercourse during the woman's ovulatory period, at which time she is most likely to conceive, and whether the male has successfully fathered healthy children with his present or any previous mate. Table 5-1 outlines the components of a thorough personal history questionnaire. In addition, a sample personal history questionnaire is shown in appendix A.

Physical Examination

A careful physical examination is critical to the fertility evaluation. This includes examination of the secondary sex characteristics (e.g., hair distribution, breast development), and of cardiovascular and neurologic function (e.g., strength of pulse in lower extremities, reflexes, pelvic sensation), as well as of the genitals. The presence and structural adequacy of the various components of the genital tract (e.g., vas deferens, prostate, epididymides) must be verified. Particular structural abnormalities associated with impaired fertility are sought (e.g., hernia, varicocele—

Table 5-1.—Patient History

Sexual history

Duration of sexual relations with and without birth control. Methods of birth control.

Sexual technique: penetration, ejaculation, use of lubricants (some are spermicidal).

Frequency and timing of coitus. Does it coincide with ovulation?

Past marital history of both partners, including pregnancies and miscarriages.

Past history: male

Developmental: age of testicular descent, age of puberty, history of prepubertal obesity, gynecomastia (excessive breast development), congenital abnormalities of urinary tract or central nervous system.

Surgical: orchiopexy (surgical placement in the scrotum of an undescended testis), pelvic or retroperitoneal (behind the abdomen) surgery, herniorrhaphy (surgical repair of a hernia), sympathectomy (interruption of sympathetic nervous system pathways), vasectomy, injury to genitals, spinal cord injury.

Medical: urinary infections, venereal disease (including nonspecific urethritis), mumps, renal disease, diabetes, radiotherapy, recent allergic febrile (fever-inducing) or viral illness (may affect semen quality), epididymis, tuberculosis, smallpox (causes obstructive azoospermia) or other chronic diseases, anosmia (absence of sense of smell), midline defects.

Drugs: complete list of all past and present medications. Many drugs may interfere with spermatogenesis, erection, ejaculation.

Occupation and habits: exposure to chemicals and heat, hot baths, steam baths, radiation, biological agents, physical exertion, cigarettes, alcohol, diet, other habits.

Sexual: libido, erectile capacity, ejaculatory capacity, position during coitus.

Past marital history of both partners: any offspring with other partners.

Previous infertility evaluations and treatments.

Past history: female

Developmental: age at onset of menstruation, age at development of secondary sex characteristics (e.g., breast development), congenital abnormalities of central nervous system.

Surgical: pelvic operations, appendectomy.

Medical: tuberculosis, venereal disease, endometriosis (aberrant appearance of uterine-like tissue in various locations in the pelvic region), tumors, menstrual irregularities, diabetes, other chronic diseases.

Menstrual: regularity of menstruation, length of menstrual cycle, number of days of menstrual bleed per cycle, premenstrual symptoms (e.g., pain, water retention).

Contraception: present and past methods.

Obstetrics: full-term deliveries, pregnancy complications, abortions, premature deliveries, previous infertility.

Drugs: complete list of all past and present medications.

Occupation and habits: exposure to chemicals, radiation, biological agents, physical exertion, cigarettes, alcohol, diet, other habits.

Sexual: libido, orgasm capacity, position during and after coitus.

Family history

- Blood disease
- Cancer
- Congenital defects
- Endocrine disorder
- Genetic disease
- Heart condition
- Impaired offspring
- Kidney disease
- Neurologic disorder
- Reproductive disorder

SOURCE: Adapted from: R. J. Sherins and S. S. Howards, "Male Infertility," *Campbell's Urology*, J. Hartwell Harrison, Reuben F. Gittes, Alan D. Perlmutter, Thomas A. Stanley, and Patrick C. Walsh (eds.) (Philadelphia: W. B. Saunders Co., 1978), pp. 715-776; and J. R. Wilson, "Infertility," *Obstetrics and Gynecology*, William J. Ledger (ed.) (St. Louis, MO: C. V. Mosby Co., 1983), pp. 160-174.

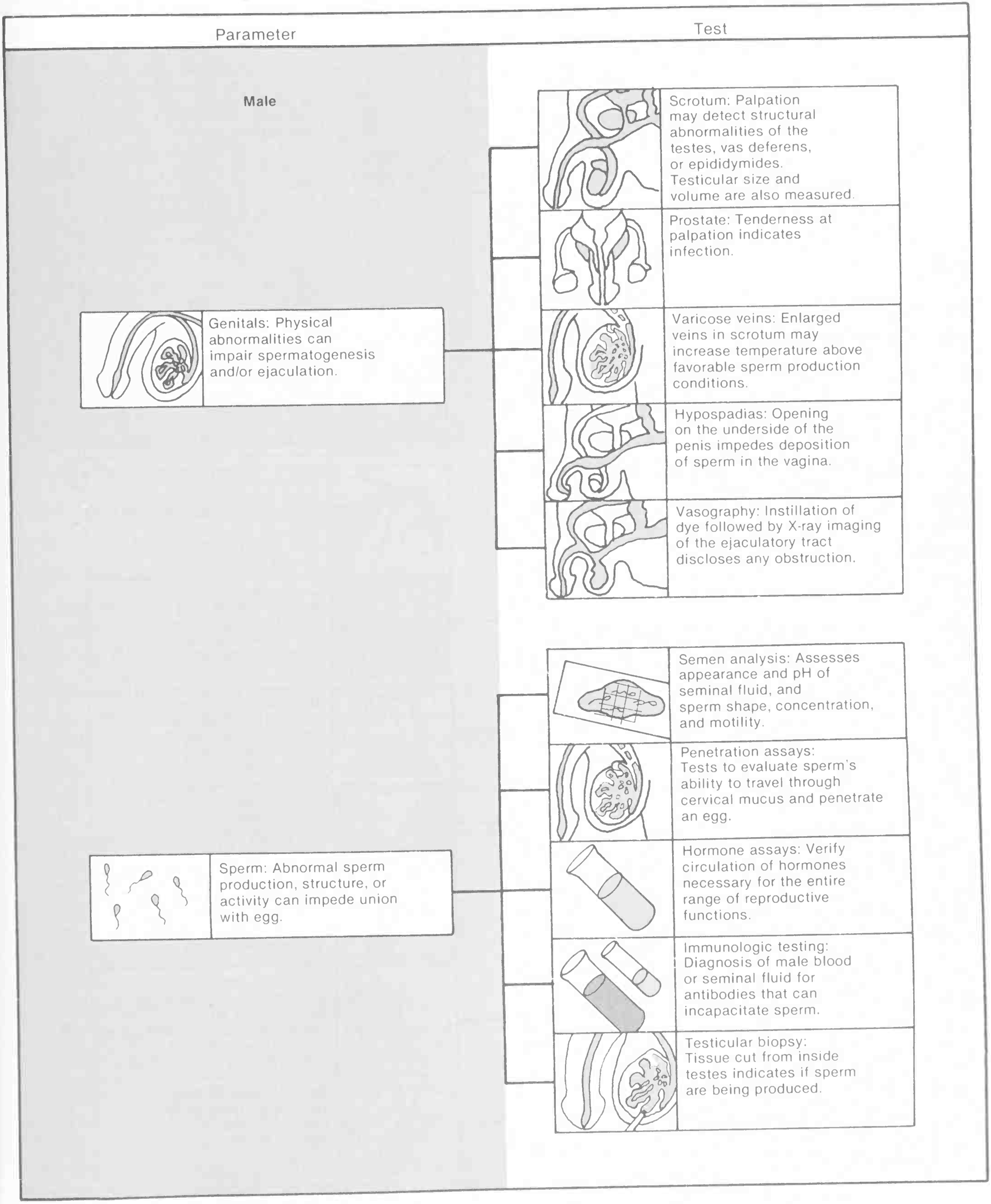
varicose veins in the testes, hypospadias—opening of the penis on the underside.) In addition, the size and volume of the testes are measured, as testicular atrophy is an indication of reduced sperm supply (3,22,59). (See figure 5-2.)

Physical examination of a patient who describes problems with impotence may include an assessment of erectile capacity. Determining the occurrence of erections during sleep (nocturnal penile tumescence—NPT) is considered one of the best means for distinguishing between physiologic and psychogenic causes of sexual dysfunction (64). NPT monitoring may be done in a laboratory or at home. The principle of the monitoring device is the same in either case: a strain gauge worn around the penis indicates changes in penile circumference during sleep.* (See figure 5-3.) While monitoring devices used at home are less precise and cannot measure certain other relevant factors (e.g., duration of erection, correlation with REM sleep cycles), some physicians find that they provide a sufficient indication of nocturnal erectile function for most patients. The cost of the home monitoring device is significantly lower than that of laboratory monitoring (i.e., $15 as opposed to $1,500) (64).

---

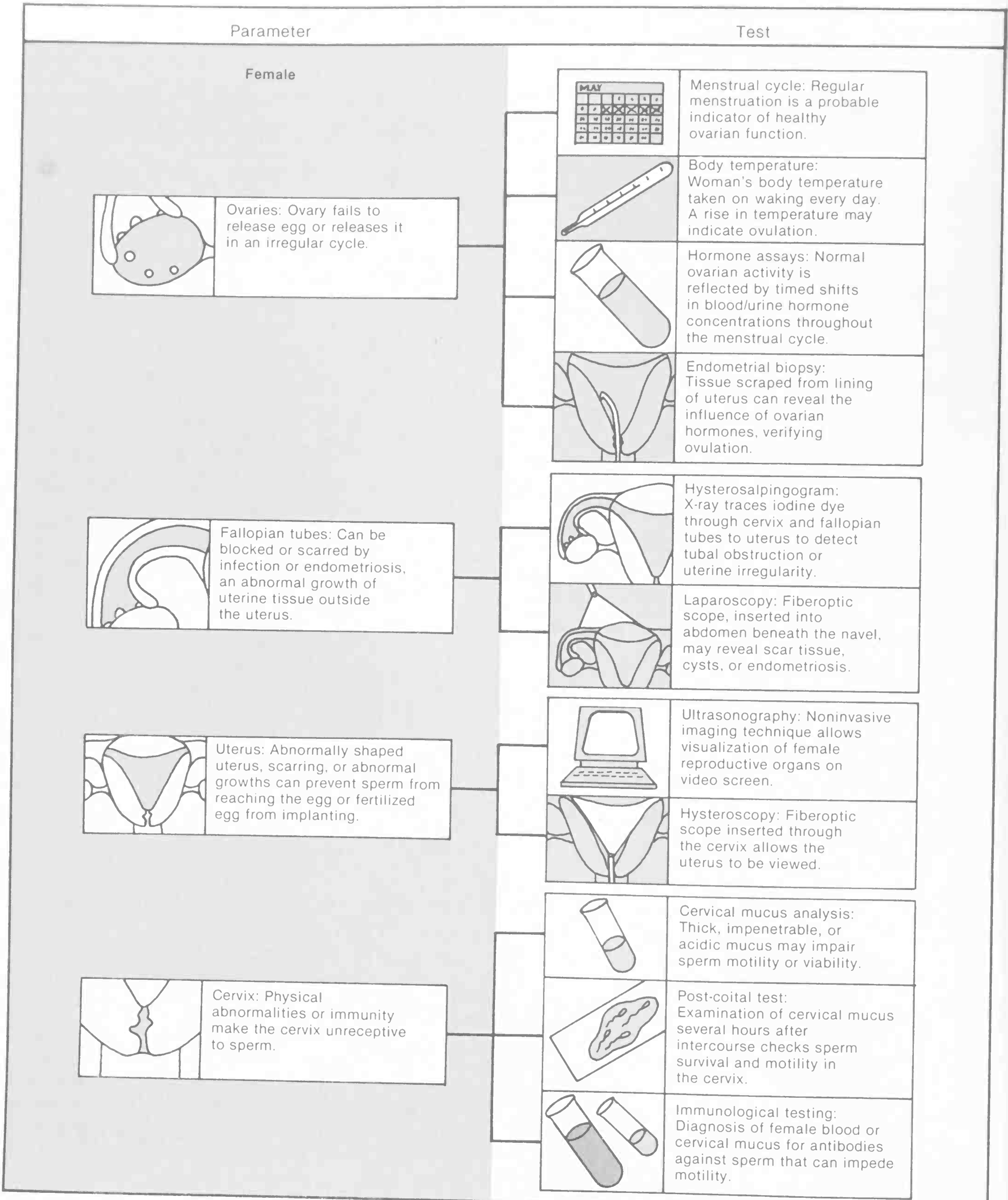
*One simple home monitoring method uses postage stamps to measure NPT. Torn perforations in a ring of stamps worn during sleep indicate nocturnal erection.

Figure 5-2.—Diagnostic Techniques in Fertility Assessment



Parameter	Test
Male	
Genitals: Physical abnormalities can impair spermatogenesis and/or ejaculation.	Scrotum: Palpation may detect structural abnormalities of the testes, vas deferens, or epididymides. Testicular size and volume are also measured. Prostate: Tenderness at palpation indicates infection. Varicose veins: Enlarged veins in scrotum may increase temperature above favorable sperm production conditions. Hypospadias: Opening on the underside of the penis impedes deposition of sperm in the vagina. Vasography: Instillation of dye followed by X-ray imaging of the ejaculatory tract discloses any obstruction.
Sperm: Abnormal sperm production, structure, or activity can impede union with egg.	Semen analysis: Assesses appearance and pH of seminal fluid, and sperm shape, concentration, and motility. Penetration assays: Tests to evaluate sperm's ability to travel through cervical mucus and penetrate an egg. Hormone assays: Verify circulation of hormones necessary for the entire range of reproductive functions. Immunologic testing: Diagnosis of male blood or seminal fluid for antibodies that can incapacitate sperm. Testicular biopsy: Tissue cut from inside testes indicates if sperm are being produced.

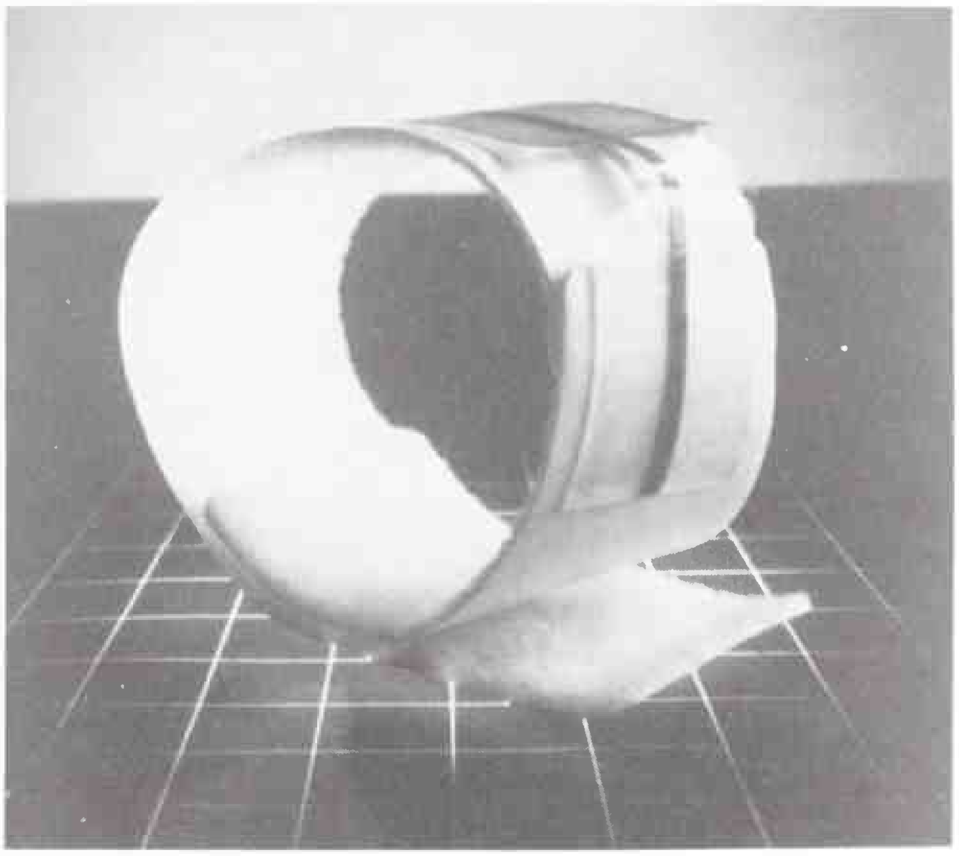
Figure 5-2.—Diagnostic Techniques in Fertility Assessment—Continued



Parameter	Test
Female	
Ovaries: Ovary fails to release egg or releases it in an irregular cycle.	Menstrual cycle: Regular menstruation is a probable indicator of healthy ovarian function.
	Body temperature: Woman's body temperature taken on waking every day. A rise in temperature may indicate ovulation.
	Hormone assays: Normal ovarian activity is reflected by timed shifts in blood/urine hormone concentrations throughout the menstrual cycle.
	Endometrial biopsy: Tissue scraped from lining of uterus can reveal the influence of ovarian hormones, verifying ovulation.
Fallopian tubes: Can be blocked or scarred by infection or endometriosis, an abnormal growth of uterine tissue outside the uterus.	Hysterosalpingogram: X-ray traces iodine dye through cervix and fallopian tubes to uterus to detect tubal obstruction or uterine irregularity.
	Laparoscopy: Fiberoptic scope, inserted into abdomen beneath the navel, may reveal scar tissue, cysts, or endometriosis.
Uterus: Abnormally shaped uterus, scarring, or abnormal growths can prevent sperm from reaching the egg or fertilized egg from implanting.	Ultrasonography: Noninvasive imaging technique allows visualization of female reproductive organs on video screen.
	Hysteroscopy: Fiberoptic scope inserted through the cervix allows the uterus to be viewed.
Cervix: Physical abnormalities or immunity make the cervix unreceptive to sperm.	Cervical mucus analysis: Thick, impenetrable, or acidic mucus may impair sperm motility or viability.
	Post-coital test: Examination of cervical mucus several hours after intercourse checks sperm survival and motility in the cervix.
	Immunological testing: Diagnosis of female blood or cervical mucus for antibodies against sperm that can impede motility.

SOURCE: Adapted by permission of M. E. Challinor and Susan Arritt for *Science 85 Magazine*. Copyright 1985 by the American Association for the Advancement of Science.

Figure 5-3.—A Gauge Used to Measure the Occurrence of Erection During Sleep



Diameter of gauge is approximately 1.5 inches

SOURCE: *Medical World News* 25:47-52, 1984.

Laboratory Evaluation

Examination of the male experiencing reproductive difficulties may include one or more of the following laboratory procedures:

- **Semen analysis.** Evaluation of semen is one of the cornerstones of the infertility examination. A variety of procedures can be used to assess the structural and functional characteristics of the patient's sperm and seminal fluid.
- **Hormone assay.** Where semen analysis repeatedly shows abnormalities, hormone assays may inform the physician about the source of the difficulties. (See Tech. Note 1.) The proper balance of hormones in the blood is critical to the entire range of reproductive functions.
- **Urinalysis and urine culture.** These screen for urinary tract infections or disorders that might hamper reproductive function.

Because of the prominence of semen analysis in examination of the male fertility patient, the following discussion examines the components of a standard semen analysis and their relevance to reproductive and sexual function.

Semen Quality

Several physical characteristics of semen have been associated with male reproductive competence. These include:

- ejaculate appearance,
- ejaculate pH,
- ejaculate volume,
- sperm density,
- sperm motility,
- sperm vitality, and
- sperm morphology.

Researchers in the field disagree as to which of these endpoints most significantly effects male fertility. There is presently no definitive indication that any **single** factor is the most important. Rather, it appears that they operate together in determining the reproductive competence of each individual (15,18,19).

In addition, there is no broadly accepted definition of what constitutes "normal semen." Laboratories differ in what they designate as the critical level for each semen characteristic (e.g., the number of motile or morphologically normal sperm, the rate of forward progression). Several factors contribute to these disparities:

- The quality and quantity of semen vary significantly among all men, even among fertile men.
- Each individual is subject to normal fluctuations in semen quality and quantity. Age, seasonal change, illness, and ejaculation frequency are among the factors known to induce these shifts.*
- Many of the measurements included in a semen analysis are subjective, qualitative judgments. This makes comparison of data from different laboratories and/or different clinicians difficult.
- Proper collection and diagnostic techniques are critical. Accuracy of findings may be compromised if the specimen is collected incorrectly, not analyzed promptly, or mishandled in any way.

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*Because of the fluctuations in semen quality and the potential for laboratory error, a minimum of three semen samples is usually recommended. An interval of at least 10 days between samples, with sexual abstinence for 2 to 4 days preceeding sample collection, is optimal (61).

- The methods and standards for statistical analysis of semen quality data vary, making comparison of results from different studies difficult.

Although efforts are being made to develop methods that will standardize and objectively measure these parameters, the time, expense, and amount of equipment they require are as yet beyond the means of many laboratories and clinics. Thus, while semen analysis remains an important aspect of a fertility examination, there are no absolute values associated with any of the physical characteristics that are assessed.

Ejaculate Appearance

Several physical properties of healthy semen make evaluation of ejaculate appearance an important step in the assessment of semen quality:

- Freshly ejaculated semen is a white, yellow, or gray fluid that coagulates at the time of ejaculation.
- Enzymes produced by the prostate gland cause the semen to liquify 3 to 25 minutes later. Semen viscosity is, therefore, a measure of secretory activity and enzymatic function of the prostate and seminal vesicles (61).
- A high incidence of agglutination (head-to-head, head-to-tail, or tail-to-tail clumping) among the spermatozoa in a sample may indicate the presence of infection or of antisperm antibodies in the seminal fluid. (See Tech. Note 2.) An observation of greater than 10 percent agglutination in a sample is considered abnormal (19,23). (See figure 5-4.)

Ejaculate pH

Normal semen pH is 7 to 8. A low pH may be the result of a contaminated sample or may indicate obstruction of the ejaculatory ducts (61).

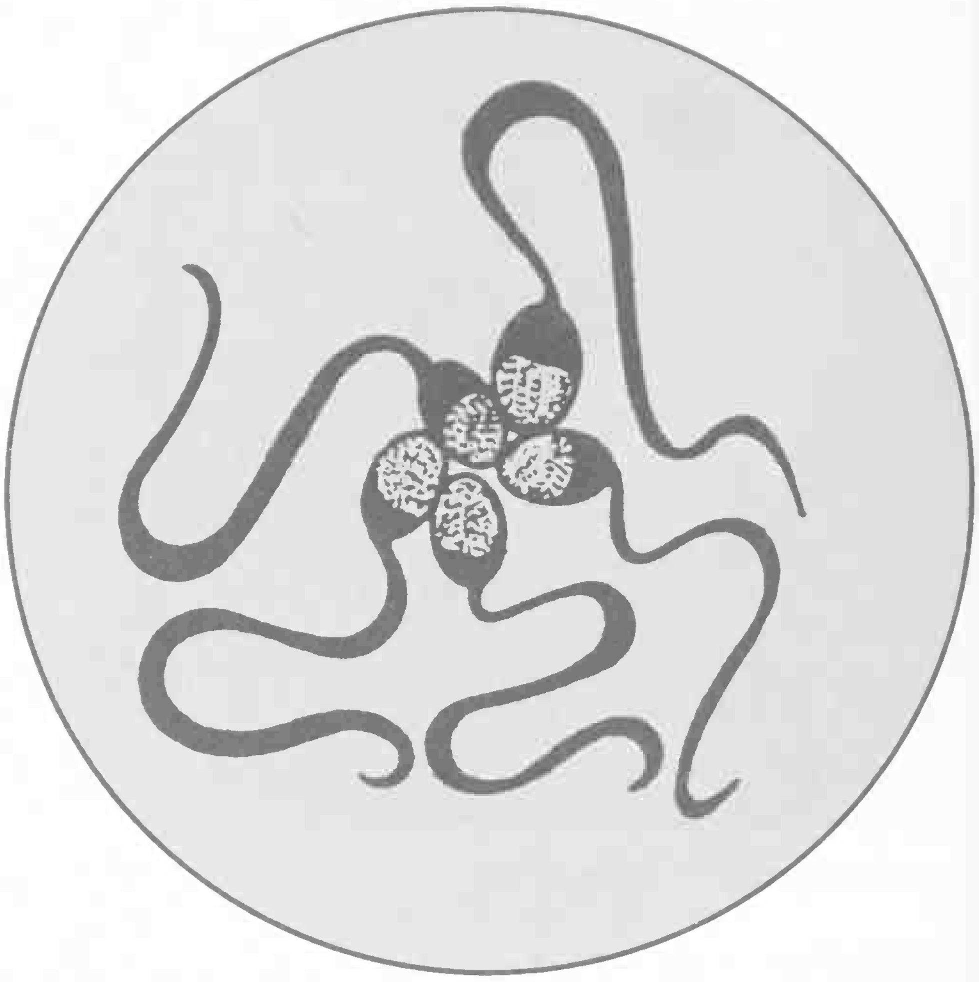
Ejaculate Volume

The amount of semen in an ejaculate normally ranges from 2.5 to 5 milliliters (19).

- Smaller volumes may indicate functional deficiencies of the prostate and/or seminal vesicles, or incomplete collection (19,23).

Figure 5-4.—Sperm Agglutination

Sperm clumping head to head (agglutination)



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- Excessive ejaculate volumes may be the result of a long period of abstinence prior to the test procedure.

Where abnormal ejaculate volumes are obtained, the test should be repeated to differentiate faulty collection technique from physiological impairment.

Sperm Density

Sperm density refers to the number of sperm per milliliter of semen. Microscopic observation enables these counts to be made. Automated techniques are also available (71).

Despite the relative ease and objectivity with which sperm density can be measured, there remains no uniformly accepted specification of the number of sperm per milliliter of semen necessary to establish fertility (23,71). (See Tech. Note 3.) Two factors contribute to this uncertainty:

1. Total semen volume and number of sperm per ejaculate differ among all men, even among fertile men. There is no sperm con-

centration threshold—except zero—below which a man is absolutely infertile.
2. Each male is subject to natural fluctuations in sperm concentration (71). Age, seasonal change, illness, and ejaculation frequency are among the factors known to induce shifts in this parameter (23).

Because of sperm density variability, at least three semen samples must be analyzed before concluding that a man is azoospermic,* oligospermic,** or normal (61). Where few or no sperm are ejaculated and normal hormone levels have been confirmed, the physician must determine whether the absence of sperm is due to impaired sperm production or to obstruction of the ejaculatory ducts. Testicular biopsy (extraction and microscopic observation of testicular tissue) and vasography (X-ray of the seminal transport system) are the two diagnostic procedures used for these purposes (59). (See Tech. Note 4.)

Sperm Motility

The importance of sperm motility in establishing male fertility is well documented (15,18,23, 33,50,70). There is a strong correlation between motile sperm and successful fertilization (7,46). No precise data on the levels of motility necessary to establish fertility are available, however, because this parameter is difficult to measure accurately and objectively (23,71).

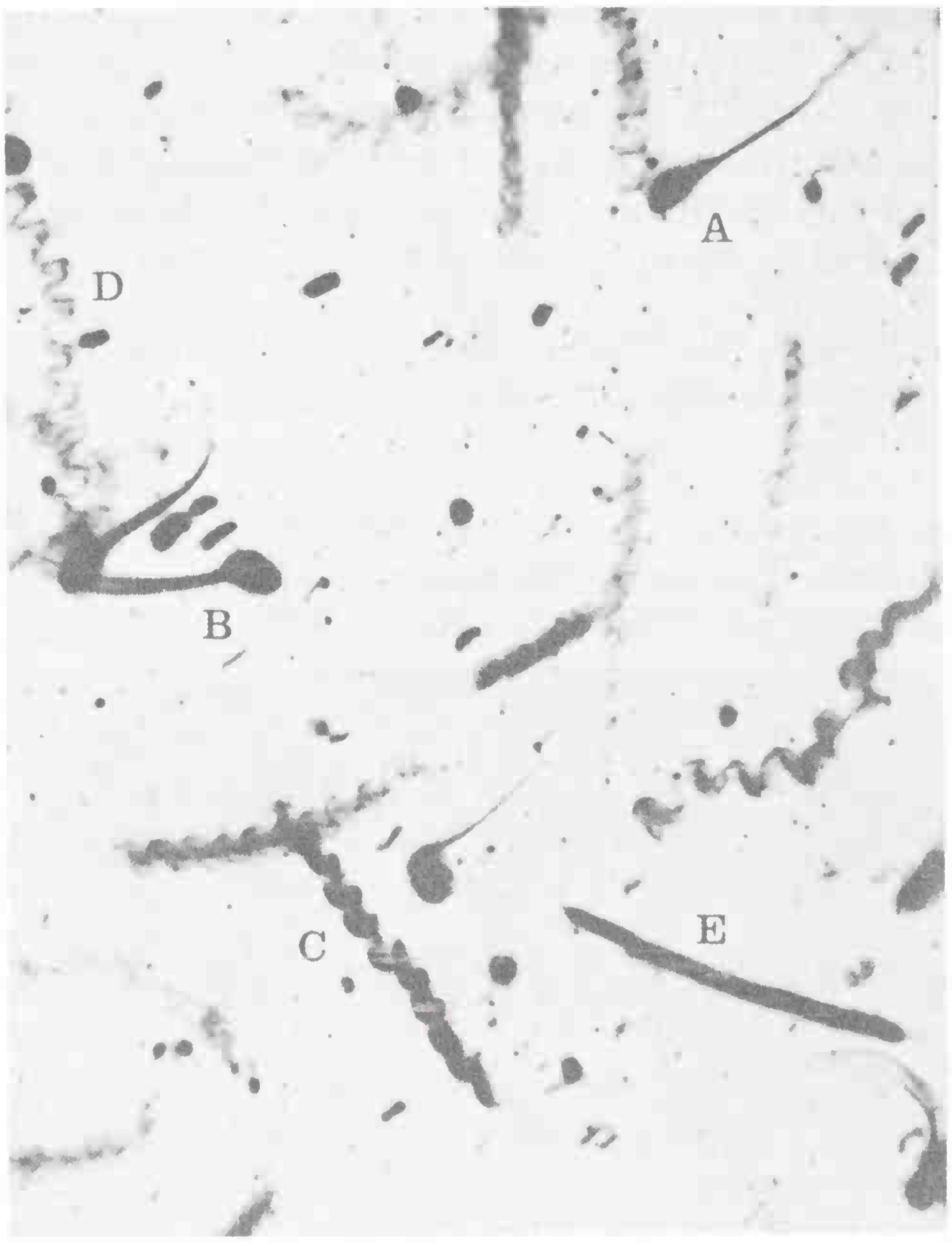
Several factors contribute to the difficulties in defining specific levels of sperm motility necessary to establish fertility:

- The sensitivity of sperm motility to temperature and to time between collection and measurement limits the comparability of data from different laboratories, where collection procedures may vary (71).
- The extreme subjectivity of the visual rating system commonly used in motility assessment makes comparison of motility data from different laboratories problematic. (See Tech. Note 5.)

Despite these difficulties, recognition of the significance of sperm motility in relation to fertility has inspired efforts to develop precise, objective measures of this parameter (23). These encompass a range of photographic and automated techniques through which overall sample motility and individual sperm velocities may be determined. (See Tech. Notes 6-8 and figure 5-5.)

While each of these techniques offers increased objectivity and accuracy in the measurement of sperm motility, the equipment, time, and expense they require limit their clinical applicability (50), and standard clinical tests of sperm motility remain of limited predictive value with regard to fertility (71).

Figure 5-5.—Sperm Movement Patterns



A) Immotile spermatozoon; B) stationary spermatozoon with active flagellum; C) rolling spermatozoon; D) yawing spermatozoon; E) straight-swimming spermatozoon exhibiting neither rolling nor yawing.

SOURCE: J. W. Overstreet, D. F. Katz, F. W. Hanson, et al., "A Simple Inexpensive Method for Objective Assessment of Human Sperm Movement Characteristics," *Fertility & Sterility* 31:162-172, 1979. Reproduced with permission of the publisher, The American Fertility Society.

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*Azoospermia is the complete absence of sperm.

**Oligospermia refers to extremely low levels of sperm production.

Sperm Vitality

A dye that selectively stains dead cells permits the ratio of live to dead spermatozoa in a sample to be determined. This technique is particularly useful in semen samples showing low levels of motility because it enables differentiation between immotile and dead sperm (19,23).

Sperm Morphology

The natural diversity of sperm shape and size among both fertile and infertile men makes it difficult to define "normal sperm morphology." While the prototypical human sperm is characterized as having an oval head, estimates of its dimensions and of the percentage of sperm that must be of this ideal morphology in order to achieve fertility are disputed by fertility experts.

The subtlety of the structural variations among sperm further complicates efforts to categorize the cells. Judgments are qualitative and subjective, limiting comparisons of morphology data from different laboratories, and making it difficult to determine the precise relationship of sperm morphology to fertility (15,18,23).

Recent efforts to standardize these measurements include the use of:

- reference slides (70);
- morphology overlays (36); and
- direct morphometric measurement (i.e., length, width, area, circumference) (35,58). (See Tech. Note 9 and figures 5-6 and 5-7.)

However, no morphology assessment technique completely eliminates the role of human decision and human error in evaluating this parameter. It remains difficult to define specific criteria for the shape, size, and percentage of "normal" sperm necessary to establish male fertility.

Despite these difficulties, there is substantial evidence for the importance of sperm morphology in establishing male reproductive capacity (36, 70).* Although subjective, evaluation of sperm morphology remains an important component of semen analysis.

Sperm Function

Sperm Function

Because tests of semen quality have failed to provide specific, reliable criteria by which to assess male reproductive capacity, researchers are

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*Studies show that morphologically abnormal sperm are poorly or nonmotile, making these misshapen cells less viable (17,36,43,52).

Figure 5-6.—Sperm Morphology: Some Categories

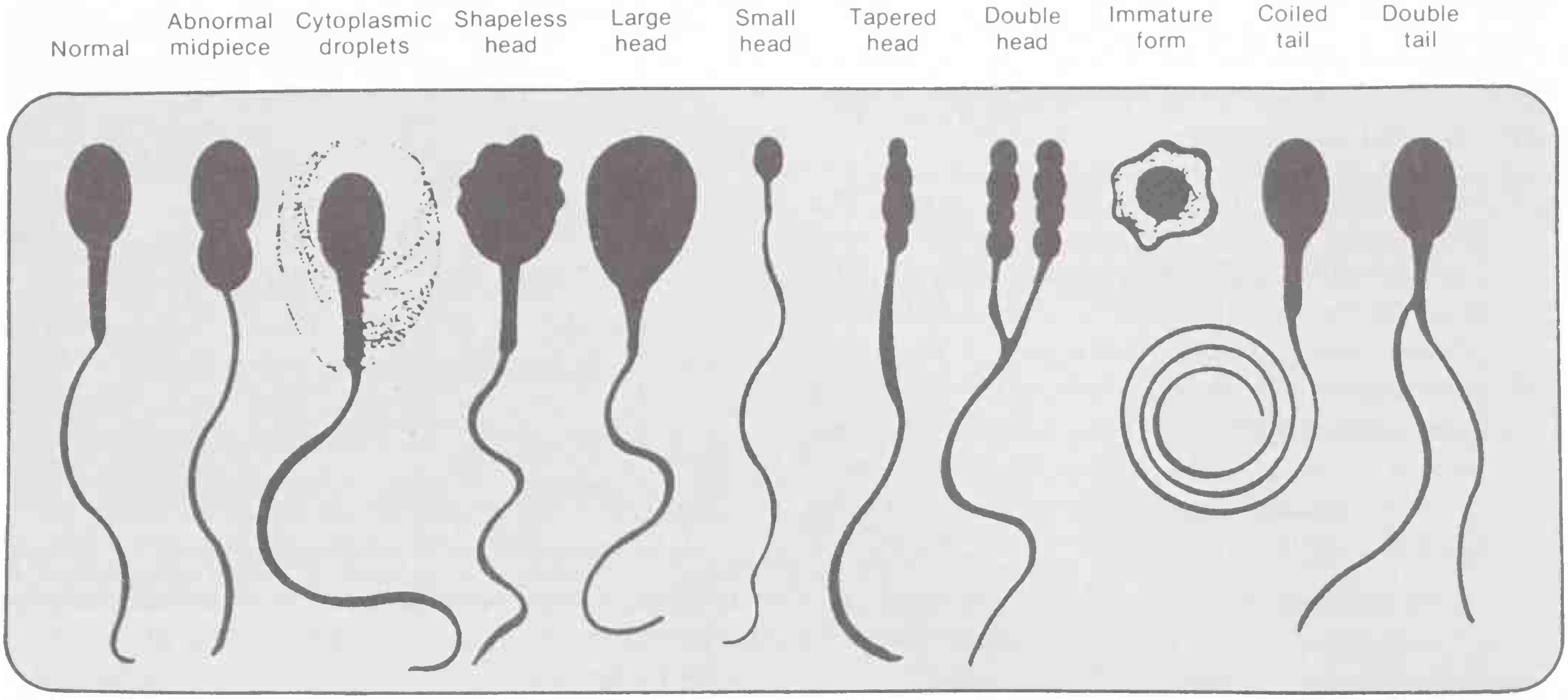

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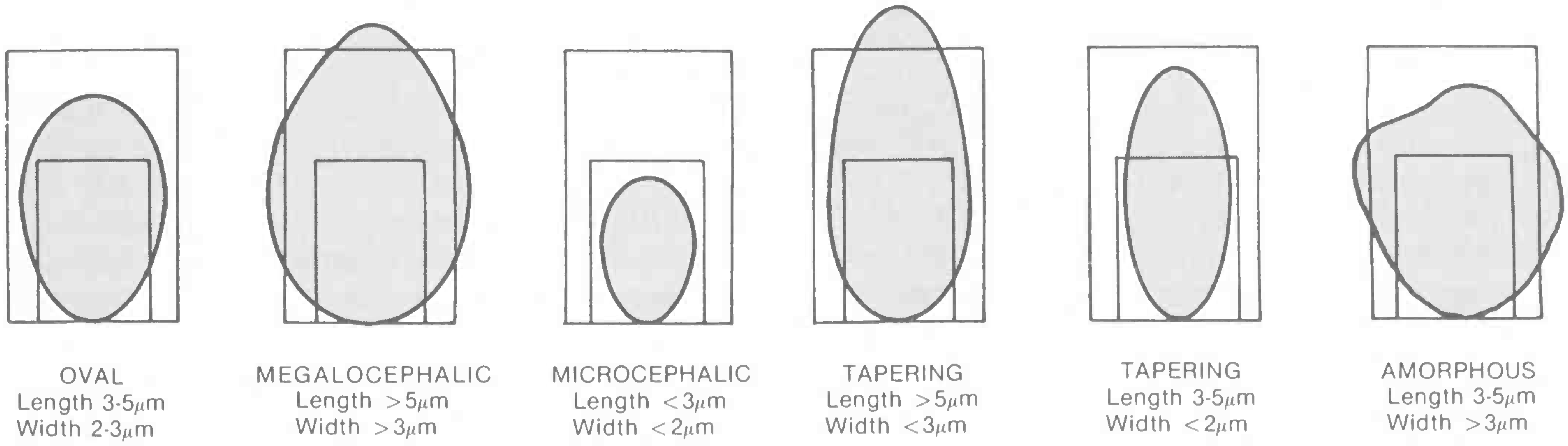
Figure 5-7.—The Morphology Overlay



Base of sperm is aligned with the bottom of the overlay. If the length and width lie between the two boxes, the classification is oval or "normal" morphology.

SOURCE: D. G. Katz, L. Diel, and J. W. Overstreet, "Differences in the Movement of Morphologically Normal and Abnormal Human Seminal Spermatozoa," *Biology of Reproduction* 26:566-570, 1982.

seeking alternative methods. These emphasize the functional ability rather than the physical characteristics of the sperm cells (1,23). While the ultimate evidence of normal sperm function is conception, the following section describes two tests that may be of predictive value.

Cervical Mucus Penetration

In order to reach and fertilize an egg cell, a sperm must migrate from the vagina through the female endocervical canal (the pathway from the vagina to the uterus). Its ability to penetrate the cervical mucus that fills this area is an important determinant in its successfully accessing the egg.

There are several laboratory techniques for the evaluation of sperm-cervical mucus interaction. (See Tech. Note 10.) Each examines the ability of the sperm to penetrate the mucus and the vitality of the sperm after penetration (i.e., some sperm may penetrate but thereafter become immobilized) (12,38,39,65).

A significant caveat of the test is the variability of cervical mucus:

- Normal changes in mucus quality occur throughout the menstrual cycle (23). As a result, a woman's mucus may resist her husband's sperm in one test, and be easily penetrated in a subsequent study (5).
- The mucus of different women, even women at the same stage of their cycle, varies in its receptivity to sperm, making it difficult to establish specific, broadly applicable criteria for mucus penetration in relation to fertility (19,23).

In order to account for these differences, it is useful to do a cross-study of both the semen and the cervical mucus with control samples. (See Tech. Note 11.) This enables the physician to determine whether the couple's fertility problem is attributable to one of the two partners or is the result of a compatibility problem.

Where a compatibility problem is suspected, it may be useful to check for the presence of antisperm antibodies. Antibodies can occur in the male—autoimmunity—or in the female—sperm allergy. They may be present in the blood serum and/or the reproductive tract of either individual and can cause varying degrees of reproductive impairment, from reduced fertility to infertility. Various techniques enable the detection of antisperm antibodies in the blood, semen, and cervical mucus. The latter presents the most difficulty, making confirmation of antisperm antibodies in the female reproductive tract problematic (9). (See Tech. Note 12.)

Sperm-Oocyte Interaction

For fertilization to occur, a single sperm cell must succeed in penetrating a female egg cell. Ethical considerations bar laboratory experimentation with human sperm penetration of **live** human eggs (1). Two alternative techniques have been developed:

1. The more common of the two techniques—the **zona-free hamster egg penetration test**—monitors the interaction of human sperm with hamster eggs (72). Its reliability as a definitive measure of male fertility, however, remains uncertain. (See Tech. Note 13.)
2. Recognizing the weaknesses of the hamster egg test, an alternative approach observes the interaction of human sperm with **non-living human eggs.** This technique is not yet widely available because of logistical difficulties in obtaining a supply of human eggs.*

Difficulty in interpreting sperm-egg interaction tests persists because the percentage of sperm that successfully penetrate a test egg varies significantly, even among fertile men. There is no universally accepted definition of what constitutes "normal sperm penetration." (See Tech. Note 13.)

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*The eggs are obtained from the ovaries of women undergoing elective surgery (8,20,49).

TESTS OF FEMALE REPRODUCTIVE HEALTH

Because of the relative inaccessibility of the female reproductive organs and their contents, assessment of female reproductive function relies heavily on inferential and indirect observations. An indirect indicator is defined here as one in which the reproductive endpoint under study is not observed, but its function is implied by the occurrence of related events. Menstrual regularity is an example. It signifies the presence of oocytes (egg cells) and the ability of the hypothalamic-pituitary-gonadal axis (the hormonal feedback system) to coordinate ovulation (the release of an egg cell from the ovary to the uterus), while none of these events is actually observed. (A discussion of female reproductive function appears in chapter 3.)

This section describes direct and indirect measures of the parameters that are assessed in a female fertility evaluation. These include:

- **Personal history:** Obtaining comprehensive information about a patient's medical, familial, occupational, and reproductive history is the first step in a fertility evaluation.
- **Secondary sex characteristics:** The attainment of pubertal milestones and normal development of secondary sex characteristics (e.g., breast development, hair distribution) are an indication of hormone secretion and response.
- **Ovarian function:** The female ovary becomes functional at the time of reproductive maturity. Events that are associated with the monthly menstrual cycle denote ovarian activity and are important indications of female reproductive health.
- **Cervical mucus:** Secretion of cervical mucus is fundamental to the female reproductive cycle. The receptivity of a woman's mucus to sperm is an important determinant of her ability to become pregnant.
- **Endometrial cells:** Accommodation of pregnancy necessitates thickening of the uterine wall. The appropriate growth response of the cells lining the uterus (endometrial cells) to monthly hormonal secretions is an important determinant of fertility.
- **Tubal patency/uterine structure:** The structural health of the fallopian tubes and uterus is necessary for the establishment of pregnancy as a fertilized egg must travel through the tubes before implanting in the uterine wall. While no methods currently enable monitoring of gamete transport, fertilization, zygote transport,or implantation, verifying the structural health of the fallopian tubes and uterus indicates the potential for these events to occur.

Assessment of these factors provides an indication of a woman's capacity to conceive. However, because the events that succeed fertilization in the reproductive process occur within the female, there are additional aspects of female reproductive competence that must be considered. These include:

- **Implantation/establishment of pregnancy:** Pregnancy is established when a fertilized egg implants in the uterine wall. While neither fertilization nor implantation are observable events, hormonal secretions provide an indication of their occurrence.
- **Embryonic differentiation and fetal development:** The ability of the female reproductive system to sustain a pregnancy and the normal development of the fetus in utero can be assessed through a variety of techniques.
- **Delivery and lactation:** These are significant aspects of female reproductive function that may be assessed to determine effects of toxic exposure.

Personal History

Obtaining information about a woman's medical, occupational, familial, and reproductive history is the critical first step in a fertility evaluation. Information obtained in this initial stage of the examination can provide important insights into the source of fertility problems. A history of menstrual irregularity, pelvic inflammatory disease, or surgery, for example, indicates a possible physiological or anatomical basis for infertility, while questions about coital frequency and technique may indicate that these are the source of the couple's inability to reproduce.

Table 5-1 lists aspects of medical, occupational, personal, and familial history that are important to a fertility assessment. The table reflects the importance of considering the infertile couple as a unit. A more detailed description of pertinent information to be obtained from the infertile couple is provided by the sample history questionnaire in appendix A.

Physical Examination

Physical examination of the fertility patient seeks evidence of physiological and/or anatomical bases for infertility. Standard health parameters (e.g., height, weight, blood pressure) and neurologic function (e.g., reflexes, pelvic sensation) are measured, and particular attention is paid to any anatomical abnormalities.

While the gonads are not external in the female as they are in the male, secondary sex characteristics (i.e., breast development, hair and fat distribution) are observable and provide an important indication of hormonal secretion and response. Excessive facial and/or body hair, for instance, may be the result of androgenization (an excess of male hormones in a female).

A standard pelvic examination, including inspection and palpation of structures throughout the genital tract, may isolate infection, tumors, adhesions, or other abnormalities contributing to reproductive difficulties.

If this initial examination fails to isolate the source of infertility, the physician undertakes a more detailed evaluation of the patient's reproductive capacity. The following section describes the specific parameters measured and the methods used. (For a summary of the diagnostic techniques used, see figure 5-2.)

Ovarian Function

Although the female genital tract and the primordial germ cells (the cells that develop into egg cells) are developed prenatally, ovarian activity first becomes apparent with the onset of menstruation at puberty. Consequently, damage sustained as a result of prenatal toxic insult may go unnoticed for the first 12 to 16 years of a girl's life (44,45).

The specifics of oocyte (egg cell) development are described in chapter 3, but it is important to note that there are fundamental differences between the production of female germ cells (eggs) and the production of male germ cells (sperm). Because of these differences, exposure to agents that are toxic to reproductive cells has different consequences for women than for men.

In a female fetus, all primordial germ cells progress to the oocyte stage before birth. There is no further generation of oocytes. An agent that is toxic to oocytes thus depletes a finite supply. A male, by contrast, continues to generate spermatogonia—the analog of the female oocyte—after he reaches reproductive maturity. From puberty onward, sperm cells are continuously produced from spermatogonia in a process that takes between 64 and 74 days. Contaminated spermatogonia are thus effectively "washed" from the system and replaced by fresh cells. This "cleansing" is obviously ineffective if exposure to the toxin continues or if there is chromosomal or hormonal damage that prevents the generation of healthy sperm (44).

Proper function of the developed ovary requires coordination of the hypothalamic-pituitary-gonadal axis; i.e., the correct balance of hormones must be present and the reproductive organs must have the capacity to respond to hormonal activity. The system relies on continual feedback mechanisms that signal increases and decreases in the production of particular hormones. Fluctuations in the concentration of these substances, in turn, cue the events of the menstrual cycle. Successful coordination of the hormonal and growth activities results in ovulation, the maturation and release of one egg cell approximately every 28 days (4,44). (See Tech. Note 14.)

Indirect Indicators

Regular Menstruation.—A regular menstrual cycle is one of the best indicators that the hypothalamic-pituitary-gonadal axis is functioning properly. Correct shifts in hormone concentration, the formation of the follicle complex (containing the oocyte to be released), the growth of this complex, and the ultimate release of the mature egg cell for passage to the uterus are all implied by the occurrence of menstruation (45). (See Tech. Note 14.)

Hormone Levels.—Hormonal feedback mechanisms are critical to ovarian function. Hormones produced outside of the ovary serve to stimulate the organ's production of additional hormonal substances. Each is necessary in order for the events in the monthly cycle (e.g., maturation and release of an egg, uterine tissue growth) to occur. Verifying the proper balance of and shifts in the levels of these substances, therefore, serves as a strong indicator of ovarian function. Blood serum and/or urine may be assayed for information on each of the relevant hormones. In addition, a new method permits levels of one key hormone, progesterone, to be monitored in saliva (74). (See Tech. Note 15 and figure 5-8.)

Basal Body Temperature.—Normal fluctuations occur in a woman's resting body temperature throughout her 28-day cycle. Basal body temperature is thus a valuable indirect measure of ovarian activity. The temperature shifts can be measured and recorded by the woman herself with a standard oral or rectal thermometer. (See Tech. Note 16, figures 5-8 and 5-9.)

Cervical Mucus.—Cervical mucus fills the cervical canal, the pathway from the vagina to the uterus. Samples can be collected quickly and easily from the cervical opening. The quality and quantity of this mucus change over the course of the menstrual cycle in accordance with estrogen fluctuations. As a result, assessment of mucus quality and quantity indicates a woman's menstrual phase. (See Tech. Note 17, figures 5-8 and 5-10.)

The changes that occur in conjunction with ovulation make it the only time of the menstrual cycle during which the mucus is penetrable by sperm. Observation of cervical mucus quality, particularly preovulatory mucus, is thus important in assessing female fertility (32,68).

Timed Endometrial Biopsy: Adequacy of Luteal Phase.—Endometrial samples (tissue from the uterine wall) provide good evidence of ovarian activity and of the adequacy (length) of the luteal phase. The luteal phase, the portion of the menstrual cycle that occurs between ovulation and menses, is characterized by a thickening of the uterine wall tissue. A typical luteal phase is precisely 14 days long. Deviation indicates a luteal phase deficiency (32,56,67). (See Tech. Note 18, figures 5-8 and 5-9.)

Timed endometrial biopsy is the standard means of identifying a luteal phase deficiency.* The procedure examines tissue from the endometrium (uterine wall). Because thickening of the endometrium is a regular occurrence of the menstrual cycle, a woman's menstrual stage may be determined based on the development of her endome-

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*Progesterone levels also indicate luteal adequacy. This hormone, secreted after ovulation, stimulates endometrial cell growth during the luteal phase. Progesterone concentrations may be monitored in blood serum and urine assays. An alternative method measures progesterone concentrations in saliva. (See Tech. Note 15a.)

Figure 5-8.—The Menstrual Cycle

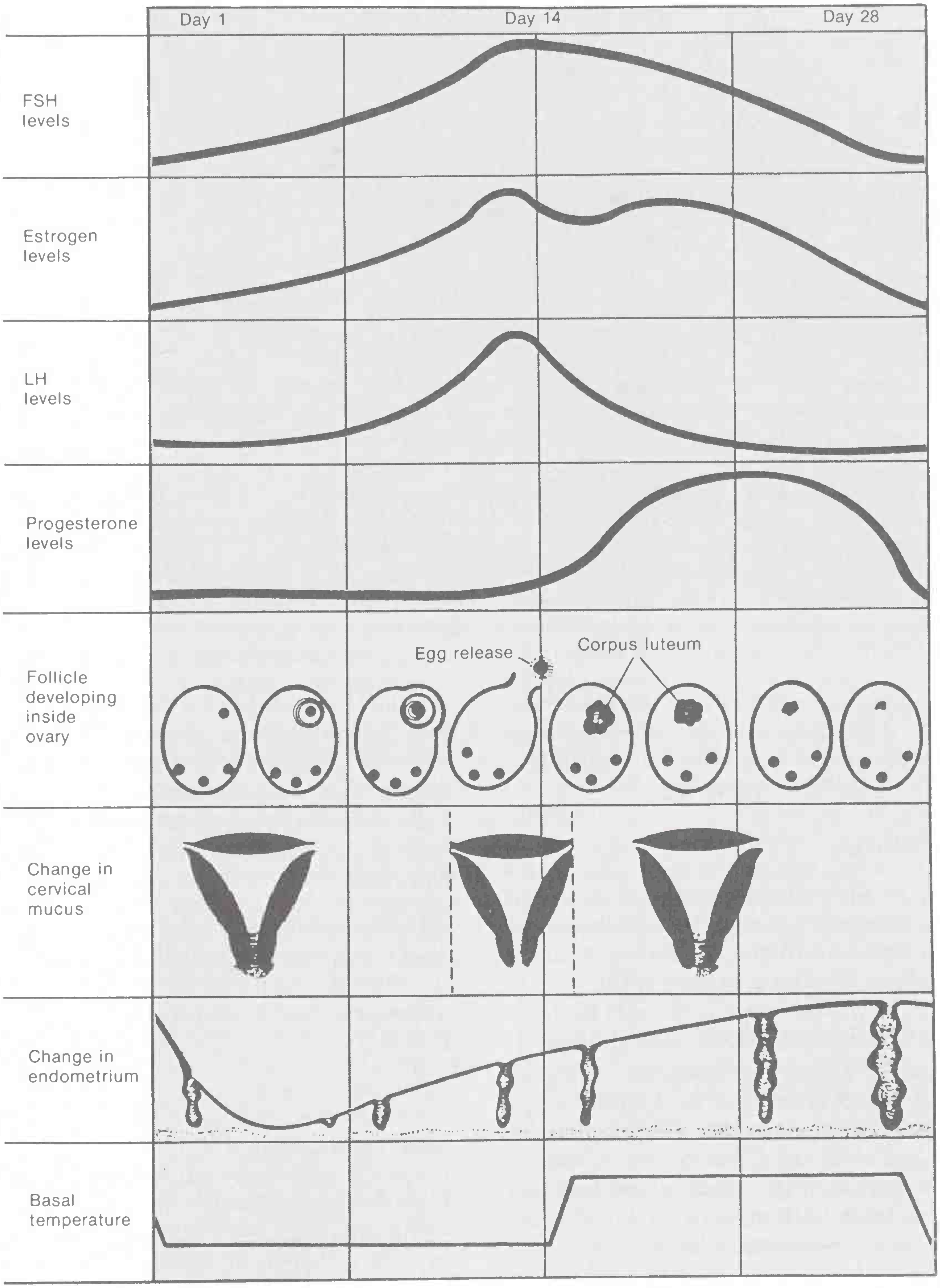
Day 1
Day 14
Day 28
FSH levels
Estrogen levels
LH levels
Progesterone levels
Follicle developing inside ovary
Egg release
Corpus luteum
Change in cervical mucus
Change in endometrium
Basal temperature

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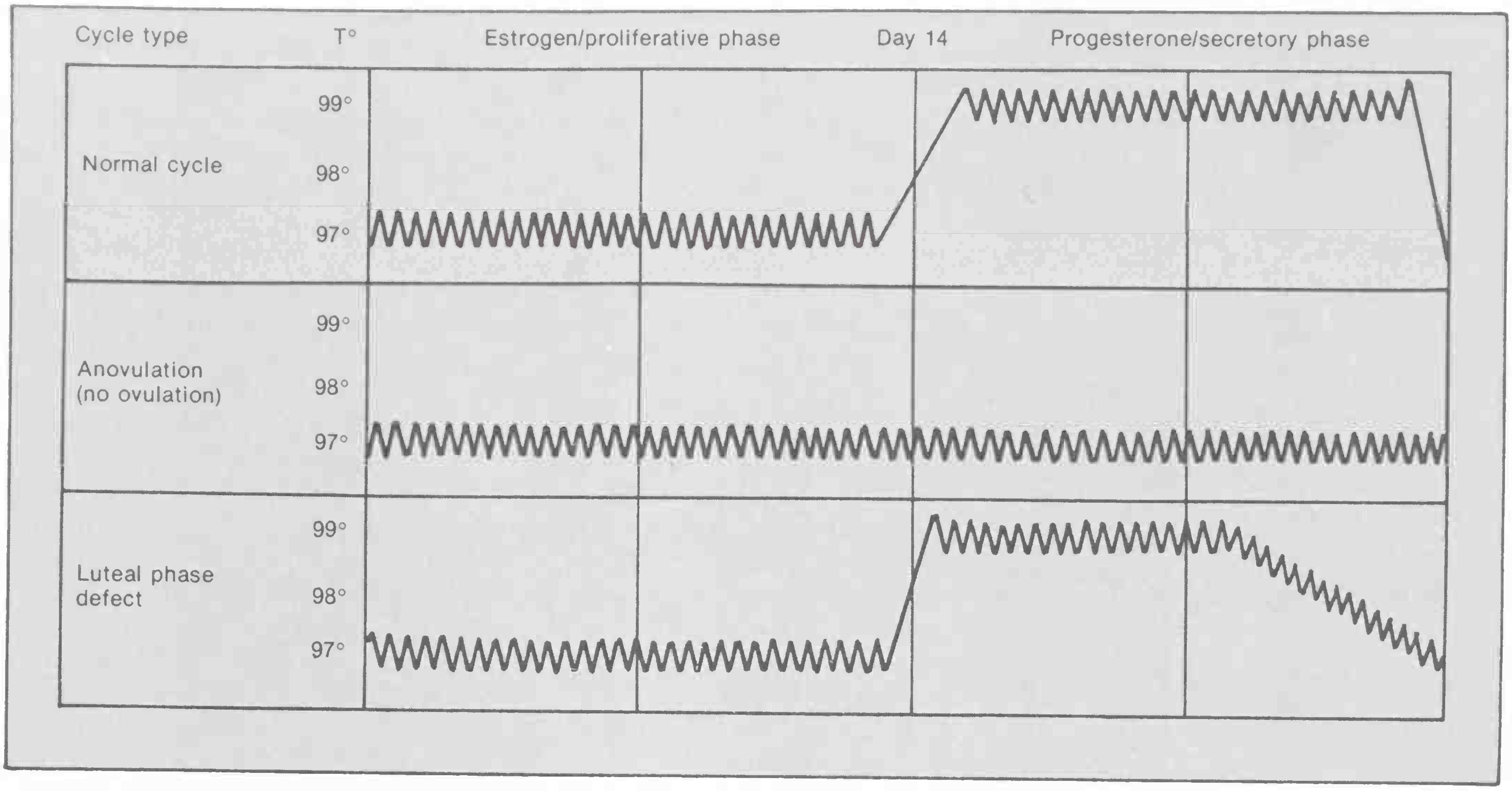
Figure 5-9.—Basal Body Temperature Patterns Throughout the Menstrual Cycle



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trial cells, and the date of her next menses may be predicted. If menses occurs sooner or later than the expected date, a luteal phase deficiency is identified (56,67). (See Tech. Note 19.)

Direct Indicators

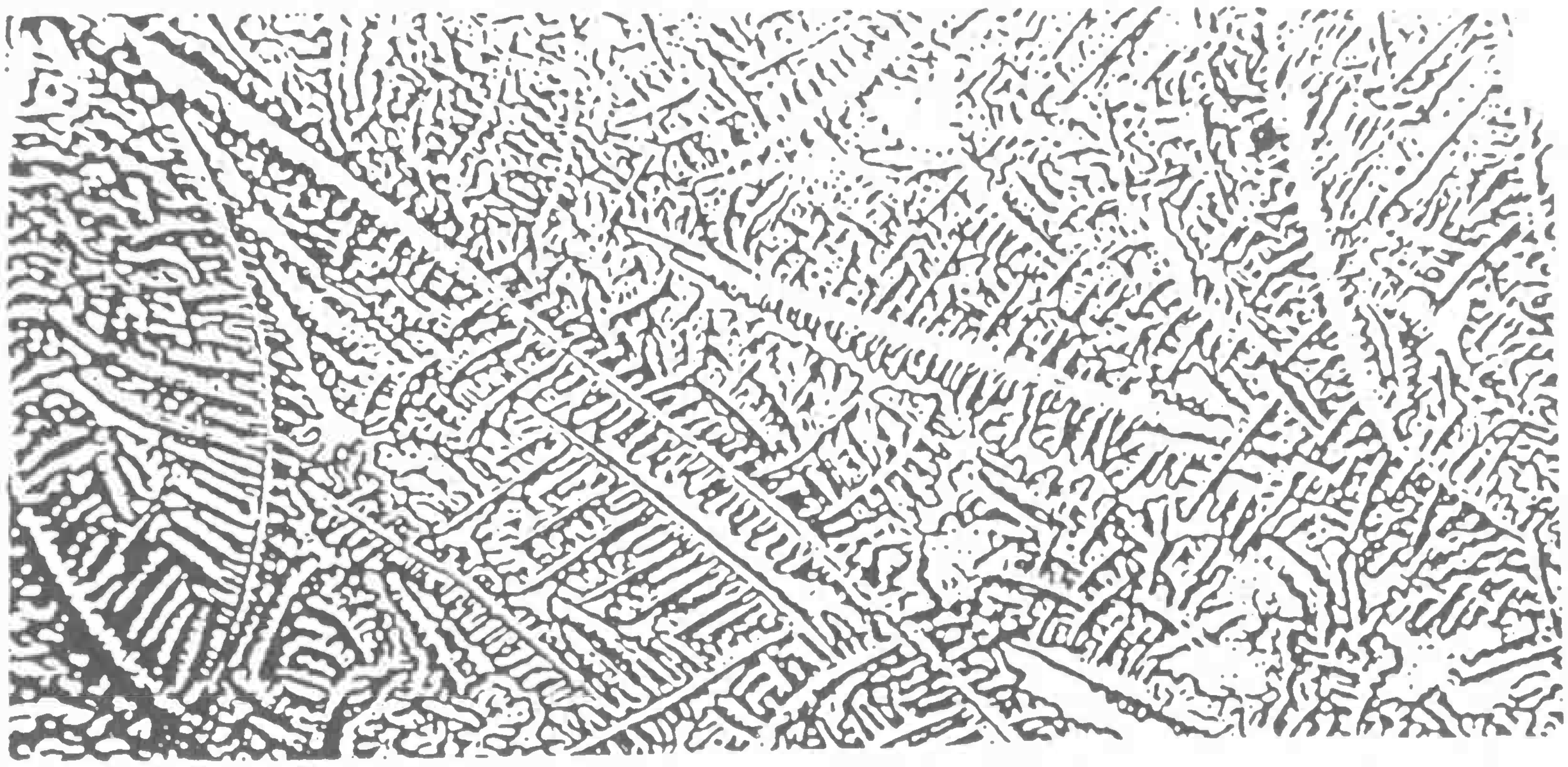
Laparoscopy.—Direct observation of an ovary to determine whether it has sustained structural damage or is deficient in oocytes requires an invasive procedure. The laparoscope is the optical instrument used to detect gross defects in ovarian structure (e.g., cysts, lesions). With the instrument inserted through a small incision in the abdominal wall, the ovaries are visible. Because it is an invasive procedure, laparoscopy is usually undertaken as a measure of last resort, when reproductive organ damage is suspected but has been unidentifiable with standard clinical diagnostic techniques. (See figure 5-1.)

Laparoscopic Ovarian Biopsy.—Observation of egg cells within the ovaries requires removal and microscopic observation of ovarian tissue. Tissue samples are taken with the laparoscope in place. While laparoscopic ovarian biopsy is a surgical procedure, it is the only means by which the contents of the ovaries can be directly observed. By viewing the tissue sample under a microscope, the presence of oocytes and of growing follicles as well as the health of the ovarian cells themselves can be verified (45). (See Tech. Note 20.)

Ultrasonography.—Ultrasonography is an imaging technique by which ovarian activity can be monitored. The projection of sonic waves into the abdominal region and the diagnosis of the wave reflections allows "visualization" of the underlying organs.

No adverse effects of ultrasonography have been demonstrated in humans. Moreover, most hospitals and many physicians have the equipment necessary for the procedure. Ultrasound imaging thus appears a safe and convenient means to monitor ovarian activity, including follicular growth and ovulation (4,12). While clinical use of ultrasound for ovarian imaging is limited, the technique is widely used as a means of fetal imaging during pregnancy. (See *Embryonic Differentiation and Fetal Development*.)

Figure 5-10.—Cervical Mucus Ferning



Just prior to ovulation, cervical mucus dries in a ferning pattern.

SOURCE: L. Speroff, R. H. Glass, and N. G. Kase, *Clinical Gynecologic Endocrinology and Infertility* (Baltimore: Williams & Wilkins Co., 1978), p. 433.

Cervical Mucus

The receptivity of cervical mucus to sperm is a critical determinant of female fertility:

- Cervical mucus quality varies in response to the hormonal shifts of the menstrual cycle, making a woman's mucus more receptive to sperm on some days than others. (See *Ovarian Function: Indirect Indicators*.)
- Certain agents may stimulate a woman's production of sperm antibodies or change the consistency or pH of her cervical mucus, making her reproductive tract unreceptive to sperm.

Evaluating the compatibility of a woman's cervical mucus with her partner's semen is thus an important measure of a couple's fertility (13).

Sperm-Cervical Mucus Interaction

Tests of sperm-cervical mucus interaction are described earlier in this chapter (see *Sperm Function*). Each examines the ability of the sperm to penetrate the mucus and sperm vitality after penetration (13,38,39). (See Tech. Note 10.)

Because of the fluctuations of both semen and cervical mucus quality, repetition of the test may be necessary to confirm results. Cross-testing of the fluids with control samples—i.e., testing the male's semen against a standard cervical mucus sample and the female's cervical mucus against a standard semen sample—is also useful. (See Tech. Note 11.)

Limited sperm motility in cervical mucus may indicate the presence of antisperm antibodies, either within the donor semen (autoimmunity) or in the reproductive tract of the female. Screening for antisperm antibodies is particularly useful where sperm motility appears poor in cervical mucus while semen analysis results are normal. (See Tech. Note 2 and Tech. Note 12.)

Endometrial Cells

The capacity of the endometrial cells (cells lining the uterus) to respond to monthly hormonal secretions is an important component of female fertility. Cyclic changes in hormone levels stimulate endometrial cell proliferation each month, preparing the uterus for pregnancy.

Endometrial Biopsy

Appropriate endometrial growth is verified through microscopic observation of endometrial tissue. Tissue is extracted by endometrial biopsy, a procedure described earlier. (See *Tests of Ovarian Function*, and Tech. Note 19.) Observation of endometrial cells also permits diagnosis of endometrial infection or disease (e.g., endometritis) that could impair fertility.

Tubal Patency/Uterine Structure

The passage of a fertilized egg through the fallopian tubes and its implantation in the uterine wall are necessary for the establishment of pregnancy. Verification of tubal patency and uterine structure are, therefore, important aspects of a female fertility evaluation.

Hysterosalpingogram

Imaging of the uterus and fallopian tubes is possible by injection of dye into the cervix and filming its spread through the peritoneal cavity. The procedure is safe and relatively painless. X-ray photography of the dye dispersion indicates any occlusion or convolution of the fallopian tubes that might prevent passage of a fertilized egg to the uterus. In addition, the size, shape, and position of the uterus and the presence of any abnormalities in the uterine wall are discernible with this technique (11,62,67).

Laparoscopy

If hysterosalpingography indicates normal tubal and uterine structure, laparoscopy may be useful. It affords the physician the opportunity for direct observation of the peritoneal cavity. The procedure is described in the discussion of direct measures of ovarian function (62).

Ultrasound

Imaging of the peritoneal cavity using ultrasound equipment may prove to be the preferred method of observation. The method is painless and, to date, does not appear to be detrimental in any way (12).

Implantation/Establishment of Pregnancy

There are no direct measures of gamete or zygote transport in humans. Consequently, the occurrence of pregnancy is the only indication that fertilization, transport, and implantation have been successfully achieved (45).

Human Chorionic Gonadotropin (hCG)

Human chorionic gonadotropin (hCG) is a substance that is secreted only during pregnancy. Blood and urine hCG assays are used to determine whether pregnancy has occurred (25,67):

- Presence of hCG in the blood serum is the earliest indication of pregnancy; i.e., it occurs before a woman misses her menstrual period (25).
- Home pregnancy detection kits screen for hCG in the urine. While the tests claim a high degree of accuracy and cost less than laboratory blood serum assays ($10 as opposed to $30), pregnancy cannot be detected as early in urine assays as it can when blood serum is used.*

Because the amount of hCG in the blood follows a specific pattern over the course of the pregnancy (see figure 5-11), monitoring hCG is also useful in detecting pregnancy loss. Sudden drastic decreases in hCG indicate that pregnancy loss has occurred. (See Tech. Note 21.)

Embryonic Differentiation and Fetal Development

In-utero monitoring of embryo/fetal development is made possible by several clinical techniques, both invasive and noninvasive. Invasive procedures sample tissue and/or fluid in attempts to diagnose systemic diseases or disorders in the developing fetus. Noninvasive fetal monitoring covers a broad range of procedures, including several imaging techniques, designed to detect structural abnormalities and/or physical manifestations of disease in the fetus or in the maternal reproductive tract. Damage to the mother or conceptus may be the result of any number of factors (e.g., exposure to one or more reproductive hazards, injury, nutritional inadequacy). By affording prenatal diagnosis of damage and/or disease, these methods contribute information that may be critical to appropriate management of pregnancy.

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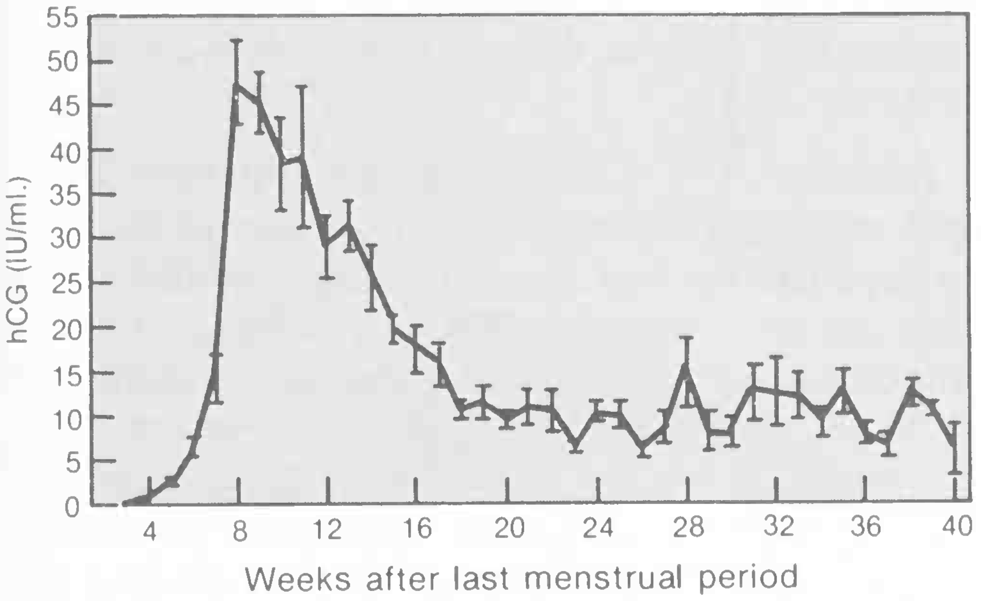
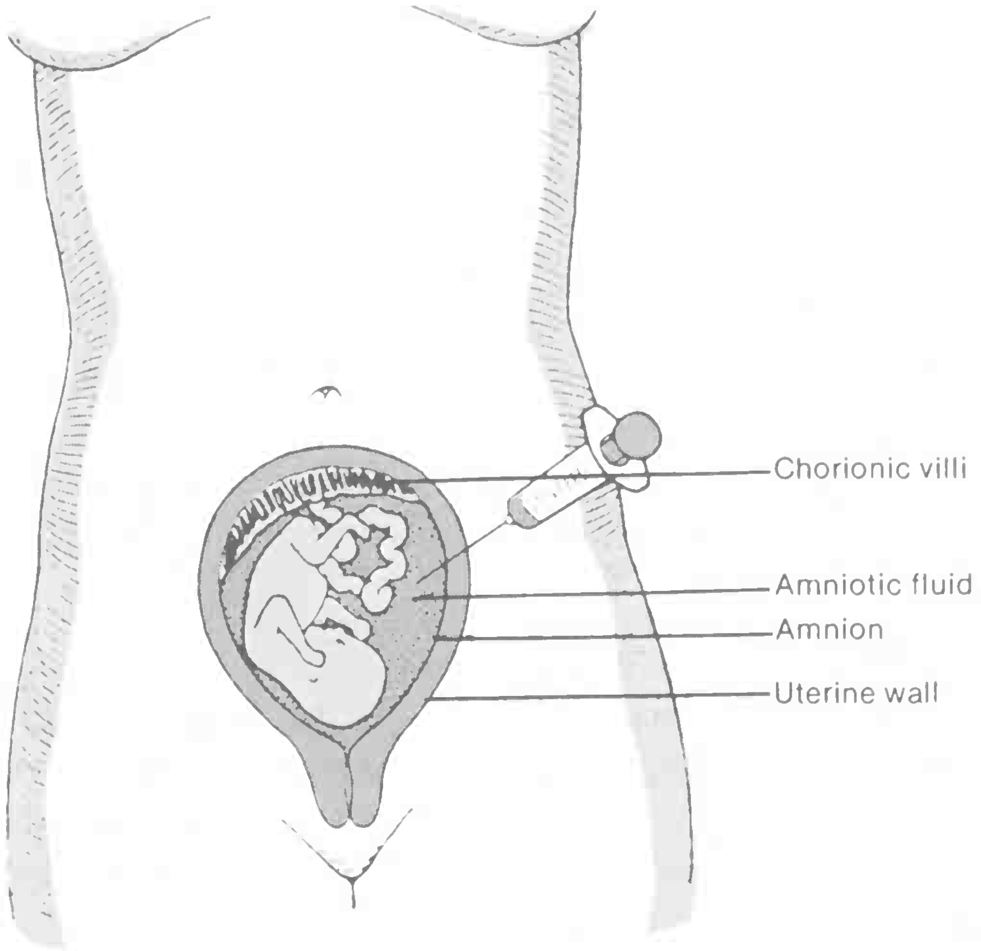
*Human chorionic gonadotropin (hCG) is apparent in the blood serum about 15 days after conception (i.e., about the time the menstrual period is expected), while it is not apparent in the urine until 4 to 6 weeks after conception.

Figure 5-11.—Mean Human Chorionic Gonadotropin (hCG) During Pregnancy



SOURCE: A.C. Wentz, J.R. Givens, R.N. Anderson, et al., *Manual of Gynecologic Endocrinology and Infertility* (Baltimore: Williams & Wilkins Co., 1979).

Figure 5-12.—Amniotic Cavity



Amniocentesis at 16 to 17 weeks.

SOURCE: Adapted from J. T. Queenan, "In Uterine Diagnosis of Down Syndrome," *Annals of NY Academy of Sciences* 171:617, 1970.

Amniocentesis

The amniotic sac is the fluid-filled cavity that surrounds the developing fetus. (See figure 5-12.) Amniocentesis is the extraction of amniotic fluid for diagnostic purposes.* The fluid contains some live cells shed by the fetus. Both the fluid itself and the cells within it provide important information about the fetus.

Amniotic cells are used primarily to diagnose chromosomal anomalies and genetic disorders:

- Disorders caused by aberrant chromosome structure or number (e.g., Down syndrome, Turner syndrome, Klinefelter syndrome) may be diagnosed by karyotyping** amniotic cells. (See Tech. Note 22.) Fetal sex is also apparent in the karyotyped cells.
- Several genetically based diseases—diseases caused by errors in the genetic information in a particular chromosome—(e.g., Tay Sachs, sickle-cell anemia, hemophilia) may be diagnosed using newly developed techniques. (See Tech. Note 23.) These are not routinely included in the analysis of amniotic cells, but are useful where specific genetic diseases are likely (e.g., one or both parents suffer from a particular hereditary disorder).***
- Enzyme and protein assays of amniotic cells may identify certain other physiological disorders in the developing fetus. (See Tech. Note 24.) These assays are generally reserved for instances in which the presence of one of these disorders is suspected.

Amniotic fluid provides additional information about fetal health:

- The fluid is most commonly assayed for the substance alpha-fetoprotein (AFP). Abnormally high levels of AFP are associated with disorders of the central nervous system, particularly neural tube defects (e.g., anencephaly, spina bifida). Elevated AFP may also reflect other systemic disorders. (See Tech. Note 25.)

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*The fluid is extracted by means of a needle that is inserted through the abdomen into the amniotic cavity.

**Karyotyping is a technique by which chromosomes are prepared for microscopic observation. It is a standard part of amniocentesis. (See Tech. Note 21.)

***Amniocentesis is considered a far safer diagnostic technique than those previously used to detect genetic disorders (e.g., fetal blood sampling to detect hemophilia, sickle-cell anemia, and other hereditary blood diseases).

- Hormone assays of the amniotic fluid are not a routine part of amniocentesis, but may be useful in diagnosing certain hormonal disorders in the fetus. (See Tech. Note 26.)

Amniocentesis is usually performed about mid-pregnancy (see Tech. Note 27) and is believed to involve a risk to the fetus of less than 0.5 percent (31). It has become relatively standard in the United States to offer the procedure to pregnant women over age 35* and to those at risk for certain fetal abnormalities. Table 5-2 outlines the situations for which amniocentesis is recommended.

Table 5-2.—Circumstances for Which Amniocentesis is Recommended

1. Pregnancies in women 35 years of age or older.
2. A previous pregnancy resulting in the birth of a chromosomally abnormal offspring.
3. Chromosomal abnormality in either parent, including:
    a. balanced translocation[a] carrier state
    b. aneupoloidy[b]
    c. mosaicism[c]
4. Down syndrome or other chromosomal abnormality in a close family member.
5. Pregnancy after three or more spontaneous abortions.
6. A previous infant born with multiple major malformations on whom no cytogenetic study was performed.
7. Fetal sex determination in pregnancies at risk of a serious X-linked hereditary disorder.
8. Biochemical studies in pregnancies at risk of a serious autosomal or X-linked recessive disorder.
9. A previous child or a parent with a neural tube defect or routine screening finds maternal serum alpha-fetoprotein level to be abnormally high.
10. Confirmation of certain abnormalities noted in a sonogram.

[a] The shifting of a segment of one chromosome into another chromosome that does not result in any excess or lost genetic material.

[b] Any deviation from the correct number of chromosomes.

[c] The presence in an individual of two distinct cell lines for a single characteristic (e.g., two blood types).

SOURCE: Adapted from: J. A. Pritchard, P. C. MacDonald, and N. F. Grant, "Techniques to Evaluate Fetal Health," *Williams Obstetrics* (Norwalk, CT: Appleton-Century-Crofts, 1985), pp. 267-293.

Fetoscopy

The fetoscope is an optical instrument that allows direct observation of the fetus. Fetoscopy is an invasive procedure, like amniocentesis, but it presents a higher level of risk to both the pregnant woman and the fetus because the instrument is much larger and remains inserted for 15 to 45 minutes (42). The procedure is associated with a risk to the fetus of approximately 20 percent. Consequently, clinical use of this technique is extremely rare.

Nonetheless, fetoscopy can provide some information that amniocentesis and other diagnostic procedures cannot. Several congenital disorders that are not detectable through analysis of amniotic fluid and cells, for example, can be identified through fetoscopy, which allows direct sampling of fetal blood and/or tissue (24). Tissue samples may identify the presence of disease in the biopsied organ, while analysis of fetal blood may detect hemophilia or various hemoglobinopathies (deficiencies of the hemoglobin) (42).

The three uses for fetoscopy include:

1. viewing the fetus,
2. sampling fetal blood and/or tissue, and
3. in-utero therapy.

Because noninvasive imaging techniques (e.g., ultrasound) exist and appear to be safer, fetoscopy is rarely used where observation of the fetus is the sole aim. (See Tech. Note 28.)

Chorionic Villus Biopsy

Chorionic villus biopsy is a method of prenatal monitoring that permits early identification of various disorders, particularly genetically based diseases (e.g., hemophilia, sickle-cell anemia). The chorion (the membrane that encases the amniotic sac containing the developing fetus) is comprised of cells derived from, and thus genetically identical to, the fetal cells (57). (See figure 5-13.)

Analysis of chorionic tissue provides the same information as amniotic fluid and cells (53). The important advantage of chorionic sampling is that it can be done much earlier in pregnancy than amniocentesis or biopsy of other fetal tissues. Chorionic villus biopsy is, in fact, the only method for diagnosis of genetic disorders that can be performed in the first trimester of pregnancy. Both amniocentesis and fetoscopy require that the fetus be in at least the second trimester of gestation (57).

The degree of risk posed by the procedure is uncertain. Preliminary data indicate a high rate

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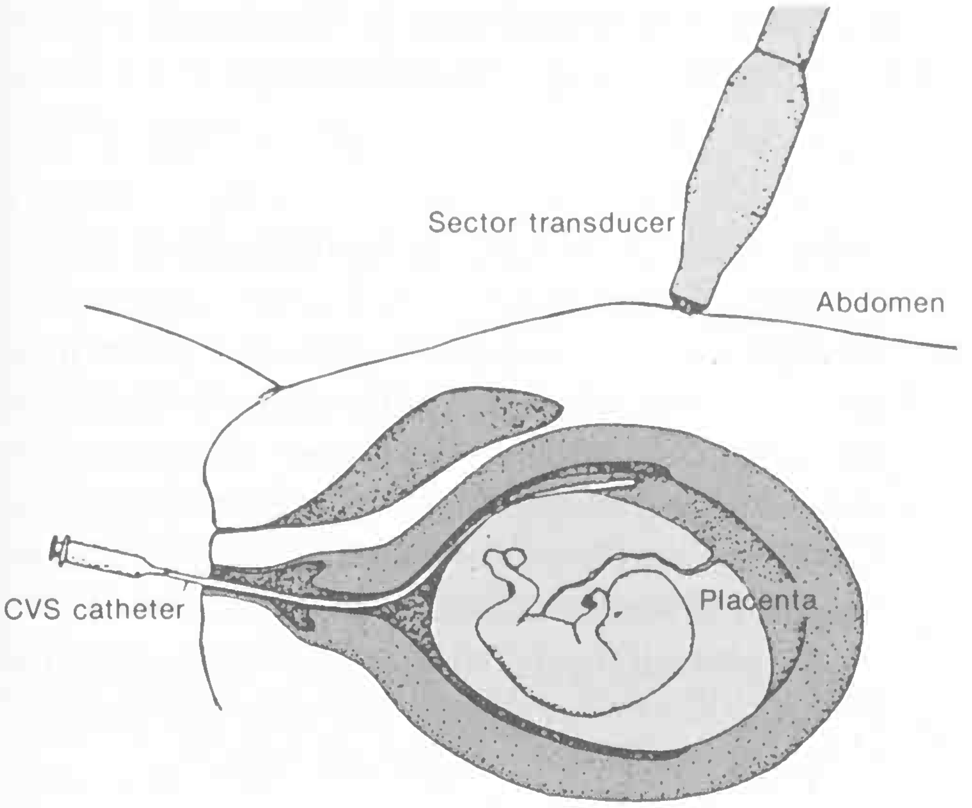
*Amniocentesis is advised for women who become pregnant during or after their mid-30s because there is an increased risk of Down syndrome associated with advanced maternal age.

Figure 5-13.—Chorionic Villus Biopsy



Diagrammatic illustration of the ultrasonically guided transcervical approach to chorionic villi sampling.

SOURCE: T. B. Perry, M. J. J. Vekemans, A. Lippman, et al., "Chorionic Villi Sampling: Clinical Experience, Immediate Complications and Patient Attitudes," *American Journal of Obstetrics and Gynecology* 151:161-166, 1985.

(12 percent) of fetal loss following chorionic villus biopsy (27). However, because the procedure is performed during the first trimester, during which time a high incidence of spontaneous abortion is normal, the post-biopsy losses may be attributable to normal early fetal loss rather than to the procedure itself (27,57). Since the actual degree of risk associated with chorionic biopsy has not been established, those who choose to undergo the test must weigh the limited information about its dangers against the advantage of having a first-trimester prenatal diagnosis (54).

Ultrasonography

Ultrasound, described earlier in conjunction with assessment of ovarian and uterine activity, is an extremely useful method of analyzing embryo/fetal development. The procedure relies on differences in acoustic densities for information about the status of the uterus and its contents. To date, no adverse effects in humans have been found to be caused by ultrasonography (12). Consequently, it has largely replaced the use of X-ray in obstetrics (53).

Sonographic Imaging.—Over the course of pregnancy, ultrasound imaging affords a vast range of diagnostic possibilities:

- Early use of ultrasound can detect ectopic pregnancies and assess gestational age. (See Tech. Note 29.)
- Beginning the seventh week of gestation, ultrasound imaging enables the embryonal heartbeat to be "visualized."
- In the second trimester, ultrasound allows detection of gross fetal malformations (e.g., anencephalies), multiple pregnancies, placental localization, progression of fetal growth (26,31,55).
- In the late stages of pregnancy, ultrasonography is useful for monitoring fetal breathing, trunk and limb movement, filling and emptying of the bladder, and quantity of amniotic fluid (53).
- Ultrasound imaging equipment may be used in conjunction with other fetal diagnostic methods; e.g., to ensure proper placement of the needle in amniocentesis.
- Ultrasound imaging facilitates delivery of a fetus whose presenting part cannot be adequately determined during labor.

Table 5-3 provides a more detailed description of the range of uses for ultrasound in obstetrics.

Its safety and potential for identifying fetal abnormalities and for providing reassurance of fetal well-being make ultrasonography an attractive diagnostic technique. In parts of Western Europe and Scandinavia, ultrasonic surveillance is considered a standard component of obstetric care. The procedure is not, as yet, routine in the United States, however, partly because of its cost (approximately $125).

Monitoring the Fetal Heart Rate.—In addition to the use of diagnostic ultrasound, ultrasound equipment is routinely used to monitor fetal heart rate. (See Tech. Note 30.) Response of the fetal heart to uterine contractions and to fetal movement has been identified as an indication of fetal well-being. The hand-held ultrasound device is also used to monitor fetal heart rate during labor and delivery.

Table 5-3.—Use of Ultrasound in Fetal Monitoring

1. Very early identification of intrauterine pregnancy.
2. Determination of gestational age: permits proper timing and management of delivery.
3. Identification of multiple fetuses, including conjoined twins.
4. Demonstration of the size and the rate of growth of the amniotic sac and the embryo, and, at times, resorption or expulsion of the embryo.
5. Measurements of the fetal head, abdominal circumference, and femur (the bone that extends from the pelvis to the knee), to help identify the duration of gestation for the normal fetus or, when measured sequentially, to help identify the growth-retarded fetus.
6. Comparison of fetal head and chest or abdominal circumference to identify hydrocephaly (accumulation of fluid in the cranium), microcephaly (abnormal smallness of the head), or anencephaly (congenital absence of the cranial vault, with brain missing or drastically reduced in size).
7. Detection of fetal anomalies such as abnormal distention of the fetal bladder, ascites (accumulation of serum in the abdominal cavity), polycystic kidneys, renal agenesis (failure of kidney to form), ovarian cyst, intestinal obstruction, diaphragmatic hernia, meningomyelocele (protrusion of brain membranes and part of the spinal cord through a defect in the vertebral column), or limb defects.
8. Demonstration of hydramnios (excess amniotic fluid), or oligohydramnios (inadequate levels of amniotic fluid) by comparing the size of the fetus to the amniotic fluid surrounding the fetus.
9. Identification of the location, size, and "maturity" of the placenta.
10. Demonstration of placental abnormalities such as hydatidiform mole (pregnancy abnormality resulting in a mass of cysts resembling a bunch of grapes), and anomalies such as chorioangioma (tumor of the chorion).
11. Identification of uterine tumors or anomalous development.
12. Detection of a foreign body such as an intrauterine device, blood clot, or retained placental fragment.
13. Monitoring fetal movement, including fetal heartbeat, breathing, trunk and limb movement, bladder function.
14. Adjunct to amniocentesis: guidance of the needle to avoid damage to placenta and/or fetus.
15. Adjunct to special procedures such as fetoscopy, intrauterine transfusion, and chorionic villus biopsy.
16. Follow-up observation of fetal anomaly identified by some other method; e.g., screening for anencephaly where amniocentesis indicates elevated alpha-fetoprotein levels.
17. Determination of fetal presentation to facilitate delivery, particularly when the presenting part cannot be adequately determined in labor or the fetal presentation is variable in late pregnancy.

SOURCE: Adapted from: J. A. Pritchard, P. C. MacDonald, and N. F. Grant, "Techniques to Evaluate Fetal Health," *Williams Obstetrics* (Norwalk, CT: Appleton-Century-Crofts, 1985), pp. 267-293; and Consensus Conference, "The Use of Diagnostic Ultrasound Imaging During Pregnancy," *Journal of the American Medical Association* 252:669-672, 1984.

X-Ray Radiography

Use of diagnostic radiography in obstetrics has become limited for several reasons:

- Some evidence suggests a correlation between prenatal exposure to ionizing radiation and fetal defects (e.g., chromosomal damage, childhood cancer).
- Uncertainty regarding the effects of the radiopaque dyes used to enhance fetal imaging has raised concern (10).[*]
- Most of the measurements made radiographically (e.g., skeletal malformations, neural tube defects, gastrointestinal obstructions, fetal tumors) can also be made using ultrasound, a method for which no correlation with fetal damage has been identified (12,53).

Despite these concerns, limited use is still made of radiography in obstetrics, particularly in the third trimester of pregnancy, when evidence suggests the fetus may be least susceptible to radiologically induced defects (10,66). Pelvimetry (X-ray of the pelvic region), for example, may help to determine the need for cesarean section when a breech (bottom-first) presentation of the fetus is discovered during labor.

Nuclear Magnetic Resonance

Nuclear magnetic resonance is a method of organ and body imaging that may become an important obstetric tool once its safety during pregnancy can be established (53). Its utility for in utero observation of structure and function has already been documented (60,63).

Delivery and Lactation

Several toxic agents can affect the ease and timing of parturition. Techniques for monitoring the status of the fetus during labor and delivery, aimed at early identification and relief of fetal distress, may provide important insights into the impact of various exposures on these aspects of reproduction (45).

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[*] Visibility of the fetus is often enhanced through instillation of water-soluble (amniography) or lipid-soluble (fetography) dyes into the amniotic fluid that surrounds the fetus (10,40).

In most pregnancies, basic clinical monitoring of the fetal heart rate, frequency of uterine contractions, and rates of cervical dilation and descent of the fetus is adequate. The fetal heart rate is monitored using either a specialized stethoscope or a hand-held ultrasound device. The heart rate is measured either intermittently or continuously, with emphasis on the rate during and immediately following uterine contractions (53).

Continuous electronic monitoring of fetal heart rate and/or uterine pressure is indicated for certain conditions; e.g., maternal diabetes, previous unexplained stillbirth, induction of labor. The electronic equipment used for these procedures, however, requires invasion of the uterus and may pose some risk to the fetus (e.g., trauma, infection) (53).

Measuring fetal blood pH at regular intervals during labor and delivery also provides an indication of fetal well-being. Like electronic monitoring, however, it is reserved for specific instances because the taking of the sample may cause trauma, infection, or damage to the fetus (53).

Lactation

A woman's ability to produce and secrete milk may be adversely affected by certain toxic exposures. Competence of lactation is an important indicator of such damage (45). In addition, several substances have been found to contaminate the milk produced by women exposed to them. In such instances, chemical analysis of milk content may be necessary to verify its suitability for consumption.

Chemical Content of Milk.—Chemical analysis of milk content provides information on the presence of toxins that may pass to the infant during maternal feeding. Various chemical assay methods (e.g., gas chromatography and high pressure liquid chromatography) are available. Depending on the compounds involved, different techniques are appropriate (69). Procedure costs vary by several orders of magnitude (i.e., from $5 to $5,000) depending on the substance for which the screening is done. To date, chemical analysis of maternal milk is undertaken only when there is reason to believe that the milk source may be contaminated at levels sufficient to affect the nursing infant.

CONCLUSION

While there are several methods by which to estimate individual reproductive capacity, physical examination and laboratory analyses can only determine that a man or woman is *potentially* fertile. Fertility is a product of the specific interaction of a particular couple. Evaluation and treatment of infertility, therefore, must consider the couple as a unit.

Furthermore, a thorough assessment of reproductive capacity cannot be limited to an evaluation of reproductive organs and reproductive cells (sperm and eggs). The multitude of parameters that comprise reproductive health are inextricably related to other physiological systems. Physical examination of the fertility patient, for example, must include assessment of circulatory, endocrine, and neurologic function. Oral or written history-taking must consider a broad range of medical factors and lifestyle characteristics that may influence reproductive health. In conjunction with the appropriate laboratory analyses, these may contribute critical insights into the cause, diagnosis, and appropriate treatment of reproductive impairment.

Examination of the male fertility patient is simplified by the fact that his reproductive organs and germ cells (sperm) are readily accessible. The female correlates are not. However, while semen analysis does permit evaluation of several aspects of male reproductive function (e.g., ejaculatory capacity), and of semen quality and quantity, there remains no positive method by which to differentiate fertile and infertile sperm. Female reproductive health can be estimated through a variety of indirect indicators (e.g., menstrual regularity, hormone levels, cervical mucus properties) and direct methods (e.g., tissue biopsy, laparoscopy, ultrasound imaging). None of these, however, constitutes absolute evidence of a woman's ability to conceive or to maintain a pregnancy.

No diagnostic method, in fact, provides positive verification of individual reproductive capacity. Even techniques that consider the interaction and compatibility of a couple as a unit (e.g., sperm-cervical mucus interaction) cannot confirm their ability to generate healthy offspring. Successful reproduction is the only absolute verification of a couple's reproductive potential.

The development of additional clinical methods may advance the evaluation of infertility and the in-utero diagnosis of fetal abnormalities, but monitoring their incidence in the population will continue to be important. Changes in frequency of reproductive difficulties (e.g., infertility, frequent miscarriage, premature birth, structurally and/or functionally impaired offspring) can provide insights into their causes, thus helping to identify those factors (i.e., workplace exposures, lifestyle characteristics) that impair human reproductive capacity.

TECHNICAL NOTES

1. Leutinizing hormone (LH), follicle stimulating hormone (FSH), and testosterone are the hormones most frequently measured in the male. The proper balance of these substances in the bloodstream is critical to the entire range of reproductive functions. (See chapter 3.)

2. Antisperm antibodies in the male seminal fluid or blood serum (autoimmunity) can result in reduced fertility or infertility. Where sperm agglutination occurs with no evidence of bacterial infection, antibody testing may reveal autoimmunity. Various illnesses and surgical procedures (e.g., vasectomy, hernia repair, testicular infection, mumps, prostatitis) alert the physician to the possibility of antisperm antibodies. A simple test, combining semen, antibody-treated blood serum, and antibody compounds, detects the presence of antisperm antibodies in the semen (i.e., if it carries antisperm antibodies, sperm will adhere to the antibody-treated blood serum) (28). An alternative method uses *immunobeads*, compounds to which antisperm antibodies adhere. By suspending sperm in a solution of immunobeads, sperm to which antibodies have been bound are identifiable (9). (See Tech. Note 11.)

Antisperm antibodies can also occur in the female, causing impaired fertility. Where sperm of good quality show poor interaction with cervical mucus in the post-coital test, it is important to screen both the male and female for antisperm antibodies.

3. The first study to correlate sperm density with fertility cited 20 million sperm per milliliter of semen as the lower limit of a "normal sperm count." This finding was based on a comparison of sperm density in 1,000 fertile and 1,000 infertile men. The researcher noted that those identified as infertile frequently had sperm counts below the 20 million level (41).

Subsequent studies, however, demonstrate that pregnancy can occur even when the sperm density is well below that level. There remains no uniformly accepted specification of the number of sperm per milliliter of semen necessary to establish fertility (23,71).

4. Testicular biopsy is the surgical removal of a wedge of testicular tissue for analysis. Where normal spermatogenesis (sperm production) is occurring, microscopic observation of the tissue should disclose all stages of growth—from immature spermatocytes to mature sperm.

Vasography describes X-ray imaging of the ejaculatory tract following instillation of radio-opaque dye in order to locate any obstruction of the ejaculatory ducts (59).

5. In the visual rating system commonly used in laboratory motility assessment, semen is examined microscopically and the number of motile sperm in several areas of the microscopic slide is used to estimate the overall percentage of motile sperm in the semen. Individual sperm are often scored according to the following scale (23):

0 = No progression
1 = Weak forward progression
2 = Moderate forward progression
3 = Active forward progression

The extreme subjectivity of these ratings makes comparison of motility data from different laboratories problematic.

6. *Videomicrography.* Videomicrography is a recently developed technique that improves objectivity in assessing sperm motility. A video camera mounted on a microscope is used to record sperm activity. The distance traveled per second (swimming speed) by individual sperm is determined using a metered viewing screen. The percentage of motile sperm in the total sample can also be estimated by this method (37).

7. *Single Image Photomicrography/High Speed Cinemicrography.* Single image photography permits observation of sperm movement. Forwardly progressing sperm appear as streaks in the time-exposed photograph. (See figure 5-4.) The swimming speed of these cells is determined by the length of the "streak" in relation to the time of the photographic exposure (e.g., 25 micrometers per second) (29,43,50).

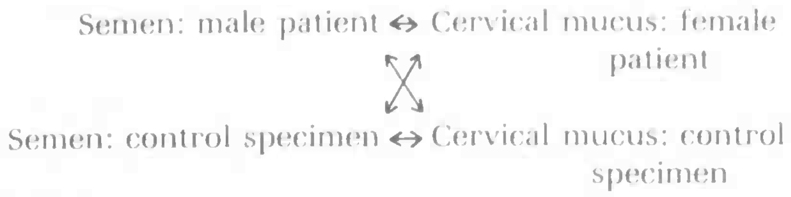
8. *Automated Analysis.* To further reduce human error and subjectivity in analysis of sperm movement, automated techniques have been introduced. These rely on computerized scanning and evaluation of photographic images to determine individual sperm velocities and the percentage of motile sperm in a sample (2,34).

9. Reference slides (70) and morphology overlays (36) are two recently developed methods that attempt to standardize morphology assessment. These establish categories (e.g., narrow head, large head, pear-shaped head) and provide standards against which to measure sperm (36,70). (See figure 5-7.)

An alternative approach is to perform direct morphometric measurement (i.e., length, width, area, circumference) of at least 50 sperm from a sample. Initial studies suggest that morphologic consistency of the sperm in a sample may correlate with fertility (35,58).

10. In the *post-coital test,* sperm are observed in a cervical mucus sample taken shortly after intercourse. An alternative method obtains separate samples of semen and cervical mucus and combines them in the laboratory to observe their interaction. Results of the post-coital sperm-cervical mucus interaction test do correlate with fertility (21).

11. The semen of the male partner can be tested against a standardized cervical mucus sample (e.g., bovine and synthetic mucus are under study); while the woman's cervical mucus can be tested for its receptivity to a semen sample of good quality. This enables the physician to determine whether the couple's fertility problem is attributable to one of the two partners or is the result of a compatibility problem.


Semen: male patient ↔ Cervical mucus: female patient
Semen: control specimen ↔ Cervical mucus: control specimen

12. Studies indicate that antisperm antibodies can be categorized according to their binding point on sperm. Those that bind to the head region appear to be most obstructive to sperm penetration of cervical mucus and/or zona pellucida of the egg cell, while tail-binding antibodies may impair sperm motility. One recently developed method enables the site of sperm-antibody binding to be identified. The technique uses immunobeads, compounds that adhere to antisperm antibodies. By suspending sperm in a solution of immunobeads, sperm to which antibodies have been bound are identifiable (9).

13. The *zona-free hamster egg penetration test* examines the ability of human sperm to penetrate hamster eggs from which the outer layer—the zona pellucida—has been removed. The zona pellucida is the major barrier to fertilization between animals of different species (1).

A substantial weakness of the test is that sperm that are able to penetrate a zona-free hamster egg may be unable to fertilize a human egg with its zona pellucida intact. The result is that men who have demonstrated fertility problems may appear normal in the zona-free hamster egg test. Studies note the occurrence of such "false positive" results (51). The test may also show "false negative" results, indicating infertility in males who have recently fathered children (19).

Studies show that penetration rates of sperm from fertile men range from 11 percent to 100 percent in sperm-egg penetration tests (1). Researchers disagree as to what constitutes "normal sperm penetration." Some identify a male as fertile if 90 percent of his sperm successfully penetrate the test egg, while others consider a single penetration an indication of reproductive competence (19).

14. There are exceptional cases, such as the event of an *anovulatory* cycle—menstruation occurring without an egg passing to the uterus. In most instances, however, regularity of menstruation is an indicator of reproductive health.

15. Assay of salivary fluids for progesterone has been suggested as an alternative to blood serum assays for this hormone. The method is particularly advantageous where serial sampling is required to monitor daily fluctuations in progesterone levels. Because adequate luteal function is reflected by ovarian secre-

tion of progesterone, the technique may also be useful in identifying luteal phase deficiency (74).

16. The increase in progesterone production that follows ovulation causes a rise of 0.5 to 1.0° in basal body temperature. These temperature shifts can be measured and recorded by the woman herself with a standard oral or rectal thermometer. Because the basal temperature reflects the lowest or resting temperature, she must take the reading immediately on waking in the morning, before arising from bed (56,67).

Although problems with the reliability of this method have been identified, many laboratories believe basal body temperature to be an extremely sensitive and accurate indicator of ovulation. They base the timing of subsequent fertility assays on the occurrence of these temperature shifts.

17. The large quantity of estrogen present immediately before ovulation stimulates increased production of cervical mucus (from 20 to 60 mg/day to 200 to 700 mg/day) (73). This mucus has particular characteristics that identify it as "preovulatory mucus." It is more watery, less viscous, and displays a "fern" drying pattern due to the crystallization of salt on the mucus filaments (see figure 5-10) (6,56,67).

18. A typical luteal phase is precisely 14 days long. Variation among women in length of menstrual cycles is usually due to differences in the number of days *preceding* ovulation while the luteal phase remains 14 days in most women. Deviation indicates a luteal phase deficiency (56,67).

19. In endometrial biopsy, uterine tissue samples are obtained by scraping the uterine wall with a small instrument inserted in the endocervical canal. Microscopic observation of the endometrial cells verifies cell proliferation in response to monthly hormonal secretions.

The degree of endometrial cell development indicates a woman's menstrual stage and allows the date of her next menses to be predicted. For example, if the endometrial tissue obtained in the biopsy shows development characteristic of the *22nd day* of the cycle, menstruation should occur 6 days later (i.e., on the *28th day*). If menses occurs sooner or later than this expected date, a luteal phase deficiency is identified (56,67).

20. The tissue samples taken in a laparoscopic ovarian biopsy represent only a minute area (0.5 centimeters) of the ovary. Other regions of the organ may vary considerably. Thus, even ovarian biopsy cannot provide a complete image of the ovary and the number of oocytes it contains.

21. Identification of pregnancy through hCG monitoring is most useful in the case of early pregnancy losses, which are otherwise difficult to detect (i.e., loss before pregnancy is visibly apparent). Some findings indicate that as many as 70 percent of all pregnancies are lost before the pregnancy itself is recognized. This is due, in part, to the amount of time that may elapse before a woman realizes that she is pregnant. Because hCG monitoring provides earlier indication of pregnancy, it could prove useful in establishing more accurate estimates of early pregnancy loss rates (16,25).

22. In karyotyped amniotic cells, numerical aberrations (more or less than the standard 46 chromosomes) as well as structural abnormalities (deleted or misplaced regions of the chromosomes) that result in abnormal formations (e.g., rings, fragments, chromosomes with obvious lesions) are detected (31). Several human disorders (e.g., Down syndrome, Turner syndrome, and Klinefelter syndrome) are known to result from these chromosomal anomalies.

23. Recently developed techniques enable a number of genetically based diseases (i.e., diseases caused by errors in the genetic information in a particular chromosome) to be diagnosed using amniotic cell chromosomes. The most common of these is a genetic mapping technique that uses enzymes (restriction endonucleases) known to cleave DNA in specific code locations. Chromosomes bearing properly coded genes yield a particular pattern of fragments when cleaved by the enzymes, while chromosomes with alternate forms of these genes are cleaved differently (14,47,48). Some diseases that are the result of a faulty gene (e.g., Tay Sachs, sickle-cell anemia, hemophilia) are identifiable with this method.

24. Enzyme and protein assays of amniotic cells are another means of diagnosing certain disorders in the developing fetus. Presence of one protein (the glial protein S-100), for example, indicates the likelihood of a central nervous system defect, while enzyme assays can detect certain metabolic disorders, such as the inability to digest specific amino acids, lipids, or sugars (31). These assays are generally reserved for instances in which the presence of one of these disorders is suspected.

25. Alpha-fetoprotein (AFP) is a protein synthesized by the fetus and present in the amniotic fluid in concentrations that decrease with gestational age. Determination of AFP levels is a standard part of amniocentesis. Abnormally high levels of AFP are associated with disorders of the central nervous system, particularly with neural tube defects (e.g., anencephaly, spina

bifida). Elevated AFP levels (greater than 20 milligrams per milliliter) may reflect other disorders, such as atresias (abnormal closures) of the digestive tube, polycystic kidneys, annular (ringlike) pancreas, hydrocephalus (accumulation of fluid in the cranium), and Fallot's tetralogy (congenital cardiac defects).

26. Proper gonadal development in the fetus requires the appropriate balance of gonadotropins and steroid hormones. The levels of these substances may be determined by analyzing the amniotic fluid (31).

27. Amniocentesis performed earlier than the 16th week often fails because of difficulties in obtaining an adequate amount of amniotic fluid and in successfully culturing the amniotic cells during the first trimester of pregnancy (24).

28. Uses of fetoscopy include:

- *Viewing the Fetus:* The small lens of the fetoscope allows detailed observation of approximately 2 to 4 square centimeters of the fetus at one time (42). This facilitates prenatal diagnosis of major external morphological malformations including facial clefts, deformed ears, limbs, and genitalia. Because noninvasive imaging techniques (e.g., ultrasound) exist and appear to be safer, fetoscopy is rarely used where observation of the fetus is the sole aim. The limited size of the fetoscope field prevents visualization of the fetus as a whole. It cannot be used to assess such things as limb size, thoracic volume, and overall anatomical symmetry (42).
- *Sampling Fetal Tissue:* The most substantial benefit that fetoscopy provides is that it permits access to fetal blood and tissue (24). Samples of the blood, skin, and/or liver tissue are taken with the fetoscope in place. Tissue samples may identify the presence of disease in the biopsied organ, while analysis of fetal blood may detect hemophilia or various hemoglobinopathies (deficiencies of the hemoglobin) (42). Further development of fetal blood assays may permit prenatal diagnosis of enzyme deficiencies, nutritional and metabolic disorders, and blood cell diseases (24).
- *Therapeutic Uses:* Development of therapeutic uses of fetoscopy, such as blood transfusions to immunodeficient fetuses, may make it a valuable method for early diagnosis and correction of fetal disorders (24). Present use of fetoscopy, however, remains limited by the level of risk posed to the developing fetus (42).

29. A delivery that is too early or too late may jeopardize the fetus. Accurate estimations of gestational age made with ultrasound are useful in determining proper timing and management of delivery (i.e., in determining the need to suppress or to induce labor).

30. In the *Contraction Stress Test,* uterine contractions are stimulated (e.g., by injection of oxytocin) and the fetal heart response monitored with ultrasound equipment. The *Nonstress Test* uses ultrasound to reflect the fetal heart response to fetal movement as identified by the mother (53).

CHAPTER 5 REFERENCES

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Chapter 6

Reproductive Risk Assessment

CONTENTS

Page

Introduction 161

The Risk Assessment Process 161
  Hazard Identification 161
  Dose-Response Assessment 162
  Exposure Assessment 162
  Risk Characterization 162

Data Used in Reproductive Risk Assessment 163
  Epidemiological Studies 163
  Toxicology Studies 166

Reproductive Research and Risk Assessment Activities in Government Agencies 171
  Environmental Protection Agency 171

National Institute for Occupational Safety and Health 174
  Reproductive Health Hazard Research 174
  Reproductive Risk Assessment 175
  Exposure Estimates 175
  Conclusions 176

Chapter 6 References 176

List of Tables

Table No. *Page*

6-1. Reproductive Endpoints for Which Population Estimates are Available 165
6-2. Sample Size Required to Detect Twofold Increase in Adverse Reproductive Outcomes 167
6-3. Tissues Separating Fetal and Maternal Blood 168
6-4. Selected Examples of Reproductive Toxic Effects Common to Animals and Humans 169
6-5. Comparison of Reported Developmental Effects of 10 Agents in Humans and in Experimental Animals 170
6-6. NIOSH Reproductive Health Hazards Research 175

Chapter 6

Reproductive Risk Assessment

INTRODUCTION

Health risk assessment is the use of scientific evidence to estimate the likelihood of adverse effects on the health of individuals or populations from specific exposures to hazardous materials and conditions. Although risk assessment is often confused with risk management, the two are different. Risk assessment attempts to evaluate the probability of occurrence of biologically significant events, while risk management determines the possible actions that can or should be taken to respond to an assessment of significant risk. This chapter discusses some of the complexities in reproductive risk assessment; risk management is the subject of chapter 7. Ethical issues surrounding the difficulty of separating value judgments from the risk assessment process are discussed in the background paper, *Ethical Issues in Reproductive Health Hazards in the Workplace*, prepared for this report (see appendix F).

Several government agencies are charged with the regulation of harmful substances and thus with risk assessment and/or risk management. A number of measures designed to centralize and standardize the risk assessment and management processes have been proposed (reviewed in ref. 5). Because these agencies have differing mandates based on the legislation underlying their authority and the types of substances and environments that are of concern, the feasibility of centralizing the risk assessment and management processes among them is uncertain. But there is the potential for establishing guidelines that can make the procedures and assumptions used in risk assessment and management processes explicit.

Health risk assessments always involve scientific uncertainties. It is not possible to predict the likelihood of a particular health effect from a given exposure situation without some degree of uncertainty regarding the exact number of people who may be affected. Scientific decisions regarding use of particular models and dose-response curves, for example, carry with them judgments that can ultimately result in different assessments of risk and thus different risk management policies. Critical steps in the risk assessment process frequently require not only scientific information, but also judgment, experience, intuition, and common sense.

THE RISK ASSESSMENT PROCESS

The risk assessment process usually contains four steps (18): hazard identification, dose-response assessment, exposure assessment, and risk characterization.

Hazard Identification

The first step in risk assessment is hazard identification, the qualitative analysis of all available animal and human data to determine whether, and at what dose, an agent is or is not likely to cause reproductive impairment. Hazard identification determines the potential of an agent to do harm, not the probability that harm will, in fact, occur (7).

Part of the task of hazard identification is to determine whether the toxin is a reproductive or developmental toxin, or both. In general, reproductive toxins are substances that affect adults. They can cause a range of effects from genetic change to systemic damage. They may act directly on reproductive organs or impair reproductive health by damaging other systems (neural, endocrine, or circulatory). Developmental toxins affect the offspring of individuals. They can cause delays in growth, malformations, cancer, behavioral changes, or death of the embryo/fetus (see chapter 3). Once the existence of a hazard has been established, the remaining steps of risk assessment—dose-response assessment, exposure assessment, and risk characterization—can begin.

Dose-Response Assessment

In dose-response assessment the relationship between the magnitude of exposure and the probability of human health effects is determined. This step nearly always involves the evaluation of animal studies that test the effects observed in a range of doses. Also involved in this process is the task of extrapolating the effects of the high doses used in animal studies to lower doses or the actual exposure levels that humans are likely to encounter. Interpretation of results is extremely complex because particular reproductive outcomes or endpoints may be difficult to observe, and numerous other variables (e.g., age, sex, lifestyle) may affect response in humans. Scientists must take account of differences in reproductive function and structure among animal species and between animals and humans; different in-utero and post-utero development; and different rates of metabolism and excretion of toxins.

Exposure Assessment

Exposure assessment identifies the population segments potentially exposed to the agent, including their composition and size as well as the magnitude, frequency, and duration of potential exposure to the agent. These data are often difficult to obtain.

Exposure to a reproductive health hazard must occur for the hazard to have an effect. Exposure may be: 1) acute (one-time) exposure, 2) episodic (recurrent but discrete) exposure, or 3) chronic (constantly present) exposure. Acute or episodic exposures are often relatively high doses over short periods of time, while chronic exposures are usually low doses over longer periods of time. Chronic exposure may also be characterized by high doses over long periods of time.

The timing and route of exposure can be very important to normal fetal development. The exposure may be of brief duration, but if it occurs at a critical point of development of the embryo/fetus, the effects can be profound. A toxin can have different effects because of the route of exposure. Some toxins have their greatest detrimental impact when inhaled. There can also be indirect exposure. The spouse, a developing embryo/fetus, or children of a worker can be exposed to substances carried home on clothing or equipment.

Reliable estimates of the number of workers potentially exposed to harmful substances and the specific substances to which they are exposed are not currently available. However, the National Institute for Occupational Safety and Health (NIOSH) is in the process of tabulating the results of an update of the 1972-74 National Occupational Hazard Survey to estimate the numbers of workers potentially exposed to specific substances. Preliminary tabulations should be available by late 1985. The information will be tabulated by sex but not by age. Estimates of exposure are extremely difficult to obtain because workers may be exposed to more than one substance and trade secrets make identification of substances difficult and time-consuming.

Estimates of human risk are complicated by individual differences in susceptibility to the effects of various levels of exposure, and the likelihood of time lag between hazard exposure and reproductive effect. Lifestyle characteristics such as smoking or alcohol consumption can increase the risk of reproductive impairment and may act additively or synergistically with hazards to which people are exposed in the workplace. Workers who have health problems associated with lower socioeconomic status may cluster in industries where hazards to their reproductive systems are more likely to be present. And people vary in their susceptibility to various harmful agents.

Risk Characterization

In this final step the data from dose-response assessment and exposure assessment are combined to estimate the actual risk from the agent. The strengths and weaknesses in each phase of the assessment are presented and summarized as a part of this step, along with the assumptions and extent of uncertainties encountered in the process. The critical component is the estimate of the level of uncertainty in the conclusions (19,23).

The transition from each step in the process is a decision point that affects allocation of resources. If the hazard assessment indicates that a hazard does not exist, resources can then be allocated to another task. If, following the risk characterization phase, a substantial risk is identified, risk management decisions must begin (see chapter 7).

DATA USED IN REPRODUCTIVE RISK ASSESSMENT

The signal that a chemical, physical, or biological agent may warrant risk assessment can come from several sources. For a new chemical, evidence may surface from toxicological tests carried out by the manufacturer in order to submit a Premanufacture Notification to the Environmental Protection Agency (EPA). However, this is an unreliable source from which to derive data on reproductive or developmental health hazards because test requirements do not specify reproductive endpoints that must be examined (28). Health hazard evaluations and NIOSH or EPA research also serve as input for risk assessments, as noted later in this chapter. Two primary sources of information are epidemiological and toxicological studies published in scientific journals.

Epidemiological Studies

Epidemiology is the study of relationships between the frequency and distribution, and the factors that may influence frequency and distribution, of diseases and injuries in human populations. The underlying tenet of epidemiology is that diseases are not distributed randomly in a population but tend to cluster (26). These groups or clusters of disease can be studied in order to discover whether the clusters are, in fact, random, or are linked to some causal factor or factors.

Epidemiology studies can have a macro or micro level of focus; both levels are important. Macro-level studies, usually surveillance systems or programs, involve large samples and are important for measuring baseline rates of reproductive endpoints such as normal and low birth weight or the frequency of congenital malformations in large segments of the population. In contrast, micro-level studies are usually concerned with a subpopulation (workers, for example) at risk because of exposure to a substance. Micro-level studies can take various forms, depending on the endpoints or group of individuals being studied.

Epidemiological studies can be divided into three broad classes: descriptive, analytical, and experimental. Descriptive and analytical studies are more often utilized for studying reproductive impairment.[1] (For further discussion of study designs see ref. 2.)

Descriptive Studies

There are two types of descriptive studies. The first, case reports (also called observational epidemiology), can highlight the occurrence of a cluster of cases of reproductive impairment, which may indicate that a potential problem exists. These are often clinical reports from occupational health physicians. The detection of infertility in DBCP-exposed men in a pesticide-manufacturing plant in California, as noted in chapter 2, is an example of this type of study. An earlier example is the detection of rubella as a causative agent of birth defects by an Australian ophthalmologist, who observed congenital cataracts in many of the offspring of his patients. When his investigations revealed that their mothers had contracted rubella during their pregnancies, he became the first to clearly implicate this disease as the cause of cataracts and other birth defects (24). This approach has two major disadvantages, however: the damage from the hazard has already occurred, and the studies are serendipitous in nature. Some hazards may thus go undetected or may already have affected large numbers of people by the time they are finally detected.

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[1] Experimental studies are difficult to undertake in industrial settings because subjects must be assigned to treatment groups. For ethical reasons, investigators must usually accept the situation as it exists with regard to exposure, and then identify appropriate comparison groups. Data from clinical trials are reviewed in the risk assessment process if they are pertinent, however. For example, results from clinical trials (experimental studies) of estrogen contraceptives are reviewed to help delineate the risk of exposure to estrogen compounds in the workplace.

The second type of study, surveillance, is important for the detection of certain kinds of reproductive dysfunction. As indicated previously, surveillance systems are usually large-scale enterprises that produce information on baseline rates in the total population. Large-scale malformation surveillance programs, for example, are an important source of information on the occurrence of birth defects. U.S. programs include the Birth Defects Monitoring Program and the Metropolitan Atlanta Congenital Defects Surveillance Program conducted by the Centers for Disease Control (CDC). (A review of State and national surveillance and monitoring programs appears in ref. 24.)

Well-designed surveillance systems have several advantages (10,24). First, they provide background incidence and prevalence rates for large numbers of persons. These background rates are valuable in detecting changes in the frequency of reproductive endpoints. Increased frequencies in time or geographical area can be checked to determine whether a true increase exists and follow-up investigations can be initiated to ascertain the cause. Second, time trends can be monitored and reproductive endpoints of specific interest can be targeted for careful investigation. Third, surveillance can provide reassurance about the absence of problems. Since the inception of birth defects surveillance programs around the world, no new teratogen has yet been initially identified in a surveillance system. Although this may indicate that the systems are not sensitive enough, most experts believe that they are adequate and that new developmental effects would have been recorded had they occurred (10,24). The major disadvantage of surveillance systems is their expense.

Micro-level concerns are the focus of monitoring studies. In these programs a population at risk can be identified and followed over time in order to detect an outcome of interest. Relatively small groups, such as persons in particular employment groups, or persons working at factories manufacturing specific products, can be studied. Monitoring systems have an advantage in that they permit observation of a population that is exposed to suspect substances. For example, a birth defects monitoring system for the Rhone-Alps region of France was able to detect an association between maternal valproic acid ingestion and the occurrence of infants born with lumbosacral neural tube defects. Valproic acid is an anticonvulsant that was used by pregnant women (3,22).

The American Petroleum Institute (10) commissioned a review of reproductive health surveillance and monitoring activities both within and outside the industry.[2] The nine U.S. oil companies that have monitoring systems have several characteristics in common: 1) reproductive monitoring is built into the existing employee health system, 2) provision is made for computer storage and editing of the data, 3) there is computer linkage to personnel records and some type of exposure data, and 4) all intend some type of analysis of this data. None have as yet analyzed the data or determined the types of statistical analyses to be used. (A summary of these systems appears in ref. 10.)

Analytical Studies

Analytical studies test for an association between exposure and outcome or result. There are three types of analytical studies: cross-sectional, case-control, and cohort. Analytical studies look for an association between an agent (e.g., exposure to a potentially harmful substance) and a particular outcome (e.g., increased rate of spontaneous abortion or lowered sperm counts). This is done by comparing a group or groups of exposed individuals with matched control groups. Cross-sectional studies compare exposed groups with control groups at one point in time; case-control studies compare individuals with a particular outcome with controls and look at prior exposure in the two groups; cohort studies follow groups that differ in amounts of exposure and look for differences in the frequency of particular outcomes in each group. (Further discussion of these studies appears in refs. 2 and 26.)

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[2] Thirty-nine companies were surveyed; 27 reported little or no activity, 3 refused to participate, and 9 agreed to be interviewed. See (10) for details.

General Considerations in Epidemiological Studies

The results of epidemiology studies may be invalid because of the complexity of factors that must be taken into consideration in the design and implementation of the studies. These factors include:

Design of the Study.—The design of the study is crucial. If the study has been improperly designed, the investigator may not be able to answer the research question or the research may take longer than necessary. Selection of the appropriate control group is also crucial. If control groups are not carefully matched with exposed groups, study results may be invalid.

Measurement of Reproductive Endpoints.—The measurement of the reproductive endpoints under study must be valid and reliable. Most reproductive endpoints are extremely difficult to measure. For example, investigators studying male infertility are not in agreement as to which tests of semen characteristics best measure infertility (validity), and test results of semen characteristics vary from laboratory to laboratory (reliability). Another endpoint, the spontaneous abortion rate, is extremely difficult to study. It has been estimated that only about 31 percent of all fertilized eggs survive to term: about 16 percent do not make the first cell division, another 15 percent are lost during the first week, and a further 27 percent during implantation. By the time of the first missed menstrual period, only about 42 percent of the fertilized eggs have survived (14,36). Many women thus spontaneously abort without realizing that they have been pregnant.

Recall bias must be considered. It is extremely difficult for all individuals to recall past events accurately.

Many reproductive endpoints are extremely rare in the population. Congenital malformations diagnosed at birth occur in about 3 percent of all births. Thus the study of a particular congenital malformation requires large numbers of births (see later discussion of sample size), and diagnoses can vary among physicians and hospitals. Many reproductive endpoints have several causes, only some of which may occur in the workplace environment.

Multiple endpoints can be affected by a particular toxicant, and there is usually no way to predict which outcomes are most likely. For example, alcohol consumption can increase the frequency of infertility, low birth weight, spontaneous abortion, congenital malformation, and developmental delay. By contrast, genetic effects may result in a variety of outcomes but show no particular pattern since genetic pathways can be affected at random (35).

The reproductive endpoints for which population frequencies are available in the United States are listed in table 6-1. No population frequencies are available for sexual dysfunction, menstrual problems, semen quality, and childhood cancer.

Table 6-1.—Reproductive Endpoints for Which Population Estimates are Available

Endpoint	Population survey[a]
1. Infertility of male and female origin	NSFG, PYS
2. Conception delay	NSFG, PYS
3. Birth rate	NSFG, NNS, NFMS, PYS
4. Pregnancy complications	NSFG, NNS, NFMS, PYS
5. Gestation at delivery (prematurity, postmaturity)	NSFG, NNS, NFMS
6. Early fetal loss (<28 weeks gestation)	NSFG, NNS, NFMS, PYS
7. Late fetal loss (>28 weeks gestation)	NSFG, NNS, NFMS, PYS
8. Sex ratio	NSFG, NNS, PYS
9. Birth weight	NSFG, NNS
10. Apgar score	NNS
11. Congenital defect	NNS
12. Infant morbidity and mortality	NSFG, NNS
13. Childhood morbidity and mortality	NNS, NFMS, PYS

[a] NSFG = 1982 National Survey of Family Growth; NNS = 1980 National Natality Survey; NFMS = 1980 National Fetal Mortality Survey; PYS = Parnes Youth Survey.

NOTE: These surveys also contain data on the following related topics: onset of menses, fertility expectations, birth spacing, contraceptive use, sterilization, care-seeking for infertility, prenatal care, spontaneous and induced abortions, maternal smoking and alcohol consumption, chronic diseases, and venereal infections in pregnancy.

SOURCE: Adapted from M. Hatch, V. Stefanchik-Scott, and Z. A. Stein, "Surveillance of Reproductive Health in the U.S.: A Survey of Activity Within and Outside Industry," unpublished, prepared for the American Petroleum Institute, December 1983.

Indications of the prevalence of some of these endpoints are available from tumor registries or individual studies from infertility and prenatal clinics (10).

Many individuals, especially workers, are reluctant to cooperate in studies because they consider them an invasion of their privacy. Some workers also believe that their medical records may be used to compromise their work status or possibilities for promotion. In addition, companies may not wish to participate in a study either because they employ their own epidemiologists or they are concerned about the liability ramifications if substances to which their employees are exposed are found to be associated with adverse effects. All of these considerations must be carefully evaluated by the investigator and must also be taken into account by those who review results of epidemiological studies during the risk assessment process.

Key Factors

The size of the sample must be adequate to demonstrate at a given level of statistical significance that there is an association between exposure and outcome variables. Three important factors are interrelated: the power of the test, the sample size needed to show a significant difference, and the presence of confounding variables.

Power.—Power is the probability of detecting a specified difference in effect between experimental and control groups. The power of a given study is determined by the sample size, background incidence of the endpoint(s) measured, and the variance of the endpoints. Power is directly related to sample size and inversely related to background incidence and variance. Power is very important because the higher the power of a test, the stronger the possible conclusions regarding the exposure-outcome relationship. If the test lacks sufficient power, two possible errors can occur:

1. the results indicate that an exposure is associated with an outcome when, in fact, there is no association (Type I error); and
2. the results show no association between the exposure and an outcome when an association in fact exists (Type II error).[3]

The probability of a Type I error is estimated with a test statistic called alpha. Before an association is said to be significant, the probability of its occurring as a result of chance sampling fluctuations (i.e., the probability of a Type I error) must be less than some predetermined value, called the statistical significance level (12,13,27).

The power is often low in studies of worker populations because the sample sizes are small. Study results, therefore, can erroneously show that exposure is not associated with the reproductive outcome when it may be.

The investigator selects the power of the test by choosing the probabilities of these two possible errors. Once this has been done, the investigator determines the frequency of the endpoint in the population in order to choose a sample of sufficient size to meet the power constraints already set (26).

Sample Size.—The adequacy of the sample size is directly related to the frequency of the reproductive endpoint in the population. If the frequency is small, for example, less than 15 percent, large samples are needed. In addition, the investigator must decide how much of a difference is a significant difference. For example, if the frequency is 15 percent, a far larger sample size would be required to show that 18 percent is a significant difference than to show that a doubling (30 percent) is a significant difference.

The frequencies of selected adverse reproductive outcomes and the sample sizes necessary to show that a twofold difference in those rates is significant are shown in table 6-2. For example, in order to detect a twofold increase in the spontaneous abortion rate (during the period from the

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[3] Type I and II errors are often defined slightly differently because the researcher is testing a null hypothesis, that is, that there is no association between two variables. The error of rejecting the null hypothesis of no association when the hypothesis is true is a Type I error. The error of not rejecting the null hypothesis when it is in fact false is a Type II error.

Table 6-2.—Sample Size Required to Detect Twofold Increase in Adverse Reproductive Outcomes[a]

Outcome	Sample size[b]
Impaired fertility:	
No conception after 1 year unprotected intercourse	322 couples
Pregnancy loss:	
Spontaneous abortion (≤20 weeks gestation)	322 pregnancies
Stillbirths	
Birth/developmental defect:	
Low birth weight	586 live births
Major birth defects (all)	631 live births
Neural tube defects	1,819 live births
Severe mental retardation	8,986 live births
Chromosomal abnormalities	17,902 live births
Infant (≤1 year) death	1,856 live births

[a] Alpha = 0.05, beta = 0.20.

[b] Divided evenly between exposed and unexposed groups.

SOURCE: M. J. Rosenberg and L. H. Kuller, "Reproductive Epidemiology: What Are the Problems in Methodology?" *Reproductive Health Policies in the Workplace*, Proceedings of Symposium held on May 10-11, 1982, Pittsburgh, PA, Family Health Council of Western Pennsylvania, Inc., 1983, pp. 201-226.

point at which a pregnancy is recognized to 20 weeks gestation), 161 pregnancies are needed in both the exposure group and the control group. In order to study this many pregnancies, the investigator must draw on a large population. Using plausible assumptions about the birth rate and number of working women, the investigator would have to draw from a population of more than 11,000 workers to find a sufficient number of pregnancies to study (24).

Confounding Factors.—A confounding factor is a variable that is correlated with both exposure and outcome. It can therefore partially or wholly account for an apparent effect of the exposure levels under study or mask an underlying true association. Confounding factors include lifestyle variables such as smoking or alcohol consumption, or ascribed characteristics such as ethnic status or age.

Maternal age, for example, can be a confounding factor. In a hypothetical study of the relationship between cumulative occupational radiation exposure and Down syndrome, the case group might contain a greater number of workers with high cumulative exposure than the control group. Because older radiation workers would be expected to have greater cumulative radiation exposure than younger workers, the risk of Down syndrome would appear to be associated with cumulative radiation exposure when it may in fact have been due to the greater age of the exposed group. In this case, maternal age would be a confounding variable since it would be associated both with the risk of Down syndrome and with cumulative radiation exposure (26).

A confounding variable that is often overlooked in studies of developmental effects is paternal exposure. If the possibility of paternally mediated effects is not considered, invalid conclusions regarding maternally mediated effects on the embryo/fetus may result.

Toxicology Studies

Toxicology studies include in vitro and whole animal tests of suspected hazards that allow the investigator to examine the roles of dose and routes of exposure. While extrapolation to humans is a complicated task, these studies, properly executed and interpreted, can *predict* an association with agents to which humans are exposed, in contrast to epidemiology studies, in which the humans will already have been affected by exposure to the hazard.

Although evidence from studies on humans is often used to refute or confirm results from animal screening tests, toxicology studies are necessary for several reasons (20):

- Experimental studies that deliberately expose humans to potentially toxic chemicals are ethically unacceptable, except in special circumstances (e.g., clinical trials for new pharmaceuticals) where there is extensive evidence from animal studies and informed consent has been given.
- Epidemiological studies of workers exposed to a chemical already in production, or reports of adverse reactions to substances, are available for only a small number of chemicals (see chapter 4).
- Even in epidemiological studies of exposed humans, results are difficult to interpret because of factors such as the lack of large enough samples and good exposure data, difficulty in measuring endpoints, and confounding variables.
- Although epidemiological studies are valuable, tests on animals have proven to be an important source of data on human risk.

Single Generation and Multigeneration Studies

Animal tests for reproductive and developmental toxicity are divided broadly into two categories: single generation studies and multigeneration studies. Single generation studies were primarily devised to test the safety of new drugs to help prevent repetition of such occurrences as the thalidomide disaster, i.e., a test of one application, usually of a high dose. Multigeneration studies were devised to test the safety of food additives and unintentional food-processing contaminants such as pesticides and packing material residues; i.e., screening for effects of chronic exposure, usually at smaller doses. These studies are conducted for two purposes:

1. to investigate mechanisms of action of toxic chemicals on various reproductive processes, and/or
2. to screen chemicals in order to identify those that may present hazards to humans exposed to them (20).

These tests are often used to evaluate the safety of chemicals before clinical trials or commercial production, sometimes without full review of their suitability as models for occupational or environmental exposures (1). (Descriptions of single generation and multigeneration study designs appear in refs. 1,4, and 20.)

General Considerations of Toxicology Studies

Design, Conduct, and Interpretation of Tests.—Evaluation of results of toxicity testing must include such considerations as the species to be selected; dosage, route, and timing of exposure; the number of animals to be used; the selection of positive and negative controls; the toxicokinetics (rates of metabolism and excretion of chemicals) of the animals being used; the endpoints under study; and whether appropriate statistical analyses have been carried out. (Discussion of these considerations appears in refs. 4 and 20.) (For discussion of experimental protocols for toxicity testing see refs. 4,11,16,17,19,21,31,32, 34,37.)

Differences in Structure and Physiology Among Animal Species and Humans.—Although reproductive processes in the mouse, rat, hamster, guinea pig, rabbit, dog, and rhesus monkey are broadly similar to those in humans, there are a number of differences in anatomy, physiology, and timing of exposure that need to be taken into account when interpreting experimental results. For example, there are substantial interspecies differences in the structure of the placenta (table 6-3). Dogs and some other species have the most tissues separating fetal and maternal blood, followed by humans and female primates, who have more than rodents and rabbits. Humans differ from experimental species in the timing and development of the placenta and in metabolism and pharmacokinetics of toxic chemicals.

The physiology of pregnancy in rodents and humans differs markedly. In rodents, for example, pituitary function is essential during the first half of the pregnancy in rodents, whereas in humans it is not required once conception has occurred (1).

Concordance Between Animals and Humans.—There are two types of concordance, that of effect and that of dose. Concordance of effect is the extent to which the types of effects observed in humans are matched by similar or related effects observed in animals, while concordance of dose is the extent to which animals and humans are affected at similar dose levels (20).

Table 6-3.—Tissues Separating Fetal and Maternal Blood

	Maternal tissue			Fetal tissue			
	Endothelium	Connective tissue	Epithelium	Trophoblast	Connective tissue	Endothelium	
Epitheliochorial	+	+	+	+	+	+	Pig, horse, donkey
Syndesmochorial	+	+	–	+	+	+	Sheep, goat, cow
Endotheliochorial	+	–	–	+	+	+	Cat, dog
Hemochorial	–	–	–	+	+	+	Woman, monkey
Hemoendothelial	–	–	–	–	–	+	Rat, rabbit, guinea pig

SOURCE: I. C. T. Nisbet and N. J. Karch, *Chemical Hazards to Human Reproduction* (Park Ridge, NJ: Noyes Data Corp., 1983), p. 94.

A basic tenet of toxicology is that effects observed in experimental animals can be used to infer likely effects (or lack of effects) in humans, with appropriate consideration of biological differences between species. And, in general, animal models do have good predictive value for humans (see chapter 4). For example, in reproductive toxicology studies, substances that affect menstrual cycles in monkeys and estrous cycles in rodents also affect menstrual cycles in humans (tables 6-4 and 6-5). Effects on fertility in rodents also seem to be a good indicator of effects in humans; most of the original work on contraceptive agents was carried out on rodents (1). However, interpreting effects of toxic doses on sexual behavior and pregnancy from animals to humans is far more complex. There are so many differences in sexual behavior between humans and animals that special care must be exercised not to misinterpret results.

Selection of the proper species is extremely important because one or even several animal species may give "false negative" results. The experience with thalidomide is a case in point. Effects similar to the phocomelic-type limb deformities observed in humans were observed in a few breeds of rabbits and seven species of primates. Thalidomide has been tested in 10 strains of rats, 15 strains of mice, 11 breeds of rabbits, 2 breeds of dogs, 3 strains of hamsters, 8 species of primates, and in cats, armadillos, guinea pigs, swine, and ferrets. Developmental effects were only occasionally produced in any of these species. However, there were fertility effects: prenatal mortality was high in rabbits, and there was a low conception rate in rats (20). This underscores the importance of selecting the appropriate species, examining other endpoints as indicators of toxic effects, and of performing human epidemiology studies to corroborate the information from ani-

Table 6-4.—Selected Examples of Reproductive Toxic Effects Common to Animals and Humans

Compound	Effect in animals	Effect in humans
Benzene	Estrous cycle disturbance: rat	Menstrual disorders
Styrene	Estrous cycle disturbance: rat	Menstrual disorders
Chlordecone (Kepone)	Testicular atrophy, decreased fertility: mouse, rat, rabbit, both sexes, females more affected	Decreased sperm count and motility, abnormal morphology
Chloroprene	Testicular damage, decreased sperm count, dominant lethal mutations: mouse, rat, cat	Decreased libido, impotence, decreased sperm count, motility, abnormal morphology. Increased spontaneous abortion in wives
DBCP	Testicular atrophy, decreased fertility, dominant lethal mutations: rat, rabbit, guinea pig	Testicular atrophy, decreased sperm count, decreased fertility
Arsenic	Embryolethal, teratogenic: mouse, hamster, rat	Low birth weight, spontaneous abortions
Carbon monoxide	Fetotoxic, low birth weight, poor postnatal development and brain damage: rodent, rabbit, sheep, pig, monkey	Fetotoxic, low birth weight, fetal brain damage
PCB	Low birth weight, high perinatal and postnatal mortality, poor postnatal growth, skin discoloration: mouse, rat, rabbit, pig, dog, monkey Prolonged estrous cycle: rat Prolonged menstrual cycle: donkey Spontaneous abortion: rhesus monkey	Low birth weight, high postnatal mortality, skin discoloration Menstrual disorders
Lead	Wide spectrum of effects: rats and mice, both sexes	Wide spectrum: both sexes
EDB	Sterility: rats, bulls	Reduced fertility in men
Carbon disulfide	Effects on spermatogenesis: rats Early embryonic mortality—increased congenital malformations: rat	Sperm abnormalities Spontaneous abortions: women

SOURCE: Adapted from S. M. Barlow and F. M. Sullivan, *Reproductive Hazards of Industrial Chemicals* (London: Academic Press, 1982), p. 16; and I. C. T. Nisbet and N. J. Karch, *Chemical Hazards to Human Reproduction* (Park Ridge, NJ: Noyes Data Corp., 1983), p. 104.

Table 6-5.—Comparison of Reported Developmental Effects of 10 Agents in Humans and in Experimental Animals

Agent	Reported sites in humans	Reported sites in animals
Anesthetic gases	Hemangiomas, hernias, skin, heart	Skeletal defects only: rat, mouse (halothane and N_2O)
Smelter emissions (lead and/or arsenic)	Multiple malformations	Multiple malformations: rat, mouse, hamster (lead and arsenic)
PBB	Skin discoloration; enlarged fontanelles	Skin discoloration and lesions: rhesus monkey; enlarged fontanelles and syndactyly: pig, dog; negative: rat, rabbit
Alcohol	Facial, CNS	Facial, dermal, neural, extremities: rat, mouse
Vinyl chloride	Neural tube	Various, including encephalocele: rat
Warfarin	Nose, bones (case reports only)	Negative: mouse, rabbit
Diphenylhydantoin	Cleft lip, cleft palate, other craniofacial, mental deficiency	Cleft lip, cleft palate, syndactyly, other skeletal defects: mouse; minor kidney anomalies: rhesus monkey
Aminopterin	Multiple malformations	Multiple malformations: sheep, rat
Busulfan	Eye, cleft palate (1 report)	Skeletal, genital defects: rat
Methotrexate	Skull, ribs, toes (2 reports)	Various: rat, cat, rabbit, mouse
Methylmercury	CNS	CNS, skeletal: rat, mouse, hamster, cat

SOURCE: I. C. T. Nisbet and N. J. Karch, *Chemical Hazards to Human Reproduction* (Park Ridge, NJ: Noyes Data Corp., 1983), pp. 97-98.

mal studies. This case also illustrates the kind of expense and level of research that may be required to determine whether substances are or are not harmful.

Dose-Response Considerations.—There is consensus among developmental biologists that thresholds do exist for the effects of toxic stimuli, unlike carcinogens (1,33). This assumption is based on biological considerations. First, the embryo has some capacity for repair of damaged tissues. Second, at early stages some systems are redundant; duplicate cells die if not used. Third, some cells have the ability to reprogram themselves. And finally, congenital abnormalities are multifactorial in nature; i.e., there is an interaction between genetic and environmental factors that determines whether an effect occurs. This can be illustrated by the action of factors causing cleft palate. Closure of the palate requires a critical balance between the size of the palatal shelves and the distance between them, which in turn depends on the width of the head and the time at which the shelves move up into the horizontal plane to fuse. If this balance is upset, either by altered tissue growth or by delay in movement of the shelves, closure of the palate may never occur (1).

In developmental toxicology testing, the assumption of threshold effects carries with it the determination of no observed effect levels (NOELs)[4] and calculation of margins of safety[5] or safety factors[6] in order to extrapolate developmental effects to humans. NOELs are difficult to establish. There is always a background rate of many of the endpoints; i.e., they occur naturally with a nonnegligible frequency. Other traits, such as the weight of an organ or birth weight, are continuously distributed. A value that represents a significant weight reduction or gain must be chosen in order to determine a NOEL. Using smaller sample sizes will yield larger NOEL values. The slope or steepness of the dose-response curve currently plays a small role in the determination of the NOEL. This curve may contain valuable information that is overlooked (6,8).

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[4] Animals are treated at three dosage levels, a high dose that produces maternal toxicity, at least one intermediate dose, and a low dose that demonstrates a NOEL. Determining a NOEL is a very complex procedure. Further discussion appears in 8, 12, 20, 22.

[5] The margin of safety approach derives a ratio of the NOEL from the most sensitive species to the estimated human exposure level from all potential sources.

[6] The safety factor approach is intended to derive a calculated exposure level that is unlikely to cause any developmental toxic responses in humans. The safety factor will vary depending on the agent, interspecies differences, and the slope of the dose-response curve. A safety factor of 100 is generally used, assuming a factor of 10 for species variability among test animals, and another 10 for animal-to-human differences. After the safety factor is selected, it is divided into the NOEL obtained from the most appropriate and/or sensitive animal species tested.

REPRODUCTIVE RESEARCH AND RISK ASSESSMENT ACTIVITIES IN GOVERNMENT AGENCIES

Discussion of reproductive research and risk assessment activities in government agencies will be confined to those of EPA, the Occupational Safety and Health Administration (OSHA), and NIOSH because this study focuses on occupational hazards. Research on reproduction in humans and toxicology testing and development of protocols, models, and guidelines is currently carried out in several government agencies.

Generally, OSHA does qualitative risk assessment for reproductive health hazards where data indicate the necessity. Risk *assessment* procedures have been made explicit in legal challenges to some standards that have been set by OSHA (see discussion in chapter 7). NIOSH, as the research and information support agency established by the OSH Act, is in the beginning phases of making risk assessment guidelines explicit, although it is carrying out research on reproductive impairment. NIOSH ranks disorders of reproduction as sixth of the 10 priority areas for research on work-related diseases and injuries (15).

EPA is currently engaged in developing guidelines for reproductive and developmental risk assessment and is also carrying out research on reproductive health hazards.

Environmental Protection Agency

Data Collection

As detailed in chapter 7, EPA obtains information on reproductive health hazards under a number of statutes. The submission requirements in most of the statutes place the burden of testing chemicals on industry. Under the Toxic Substances Control Act (TSCA), EPA receives basic data on the chemical identity of substances, their production volume, and worker exposure to the substances. The EPA Office of Toxic Substances also receives Premanufacture Notifications that help to determine the developmental (teratogenic) or mutagenic potential of proposed commercial substances. In addition, the agency receives notices when significant adverse reactions are observed in employees exposed to a substance and receives notices when substantial risks of significant environmental and health effects are observed by manufacturers.

EPA obtains data on pesticides under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). In order to collect testing information on environmental and human health effects of products not subject to recent review, EPA has implemented a program for reregistration of pesticide products "licensed" under FIFRA over the past 40 years. This program requires teratogenicity testing in two animal species (generally rats and rabbits). The program also utilizes limited means of obtaining information on adverse health effects in workers.

EPA may also collect information on reproductive health hazards as part of the Clean Air Act, the Resource Conservation and Recovery Act, the Atomic Energy Act, and possibly Superfund.

In general, however, these laws provide very little basis for the *systematic* collection of reproductive health hazard data, and virtually no regulatory authority for monitoring or collecting information on toxic occupational exposures.

Data Bases

In addition to data handling submissions, EPA participates in several independent data collection activities. The most comprehensive data base is the Chemical Substances Information Network (CSIN), which was established under TSCA and is currently maintained by EPA and the Council on Environmental Quality. CSIN's broad information base includes data on reproductive health hazards, structure, effects, uses, production, and pertinent regulatory requirements of many chemicals. Another data system, the Chemical Information System, maintained within the National Institutes of Health (NIH), contains the Scientific Parameters in Health and the Environment, which is a group of integrated data bases.

The Department of Energy's Oak Ridge National Laboratory provides internal data services on chemicals that are known or suspected reproductive health hazards via a data base called the Environmental Mutagen and Environmental Teratogen Information Center.

Internal EPA Research

The Health Effect Research Laboratory (HERL) in Research Triangle Park, North Carolina, provides research support for the Office of Research and Development's (ORD) reproductive health hazard assessments. Within HERL, the Developmental Biology Division conducts research in developmental toxicology and reproductive toxicology. For example, when there is disagreement concerning the toxicity of a particular substance being considered for regulation, the division will perform the research necessary to resolve the dispute. The division also reviews certain compounds for their reproductive effects. While the division does not perform risk assessments per se, it assesses the exposure of a compound, supplies input for risk assessment models, and makes recommendations concerning standards for a substance's continued use.

The agency also relies on CDC and FDA for research on reproductive health hazards. When specific substances are being considered for regulation, information on reproductive health hazards is exchanged under FIFRA and TSCA with OSHA and, to a more limited extent, with the Consumer Product Safety Commission (CPSC). The National Toxicology Program, under the supervision of the Public Health Service in the Department of Health and Human Services (DHHS), in which EPA is a participating agency, may also provide assistance through its coordination and monitoring of interagency research, testing, and method development.

In some limited instances, EPA may employ outside contractors to perform certain tests to provide data necessary for risk assessments being performed by the agency.

Peer Review Procedures

EPA risk assessments and the resulting regulatory decisions undergo peer review in several ways. At the request of agency officials, risk assessments performed within ORD are reviewed by professionals in the field both within and outside the Agency. Occasionally individuals in other agencies are informally requested to review ORD's risk assessment work.

The second review method for risk assessment is through internal agency procedures and informal case-by-case referrals to different program offices. These are also not mandated by any particular statute. Red-border review[7] of regulatory actions is perhaps the most visible review of risk assessments within the agency. Before any regulatory proposal is published by EPA, a regulatory package is assembled by the program office with responsibility for the action and is distributed for review and approval to each assistant administrator in EPA.

Risk assessments are also reviewed on an informal basis within EPA by intra-agency task forces formed on a case-by-case basis to review particular chemicals. Risk assessments on reproductive health hazards are also regularly referred to the Developmental Biology Division in Research Triangle Park, North Carolina. For ionizing radiation, the agency has traditionally relied on periodic reviews conducted by the National Academy of Sciences at the agency's request. Finally, risk assessments are reviewed by independent advisory groups established pursuant to the environmental statutes themselves or to the Environmental Research and Development Act.

Assessment of Reproductive Health Hazards

Under TSCA and FIFRA, the Office of Pesticide Programs (OPP) and the Office of Toxic Substances (OTS) are responsible for analyzing the industry data submitted to EPA. Risk assessments are performed in OPP by the Hazard Evaluation Division and in OTS by the Health and Environmental Review Division. These offices are staffed by toxicologists, biologists, and statisticians. Scientists working in one of these branches are sometimes unaware of work being done in their functionally equivalent branch.

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[7] "Red border review" denotes intra-agency EPA procedures for the review of all agency rulemaking proposals by all assistant administrators in EPA. The term comes from the fact that these proposed regulatory actions are routed through EPA in red folders.

Other EPA program offices do not generally conduct their own risk assessments of particular substances. They rely instead on the Office of Health and Environmental Assessment in ORD if a risk assessment is required. An exception is the Office of Radiation Programs, which maintains it own health effects staff. In ORD, the Reproductive Effects Assessment Group (REAG), staffed by 15 scientists (reproductive and developmental toxicologists, epidemiologists, pharmacologists, biologists, and geneticists), conducts reproductive risk assessments for most program offices other than OPP and OTS. They also perform some risk assessments for OPP and OTS on a case-by-case basis. OPP and OTS risk assessments are generally reviewed by the Assistant Administrator of ORD only if a regulatory action is proposed and proceeds through red-border review. This is to assure consistency of all risk assessments done by EPA.

Risk assessment procedures for reproductive health hazards, while appearing to be fairly consistent among offices, are still perceived as problematic by the agency's officials.

EPA Proposed Risk Assessment Guidelines

At the request of the former administrator, ORD is developing six specialized risk assessment guidelines: 1) mutagenicity, 2) developmental toxicology, 3) exposure, 4) carcinogenicity, 5) complex mixtures, and 6) male and female reproductive impairment. REAG has the responsibility for three: developmental toxicants, mutagens, and male/female reproductive effects.[8] REAG anticipates drafting the Male/Female Reproductive Effects Risk Assessment Guidelines by 1986.

In the developmental toxicology guidelines, EPA, for the most part, continues to recommend safety factors and margins of safety in risk assessment determinations, but acknowledges that more research needs to be done on mathematical modeling from dose-response curves. REAG and the Office of Research are currently developing methodology in this area. EPA officials expect the guidelines to be constantly revised as new advances are made in the science.

REAG staff have also been contributing developmental toxicology and reproductive toxicology guidelines to the Interagency Risk Management Council. (Member agencies include the Food and Drug Administration, the U.S. Department of Agriculture, NIH, OSHA, CPSC, and EPA). The goal of this council is to attempt the drafting of consistent policies across all executive regulatory agencies. This effort had been expected to take 2 years, but is now stalled because of a lack of resources.

Conclusions

EPA's collection of data and research on reproductive health hazards appears disjointed. Probably because of programmatic divisions within the agency, data developed under one statute are often not routinely shared with offices carrying out other statutory responsibilities. Although this may be a consequence of the fact that EPA operates under several different legislative mandates, it may inhibit regulatory consideration of chemicals with potential for reproductive effects in different exposure situations that are covered by different mandates. It may also lead to duplication of internal and external testing.

Data retrieval systems appear to offer one avenue for the coordination of this information. One system, the Status Report of Chemical Activities published through the Toxics Information Series, is a particularly useful model in this regard. The status report lists, by chemical, testing being performed on a particular substance, the statutory authority under which it is being performed, and a contact person within the agency. It also indicates whether a regulatory action is being contemplated or has been taken.

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[8] Four of the proposed guidelines were published in the Federal Register, vol. 49, No. 227, Nov. 23, 1984: Carcinogen Risk Assessment, p. 46294; Exposure Risk Assessment, p. 46304; Mutagenicity Risk Assessment, p. 46314; and Health Assessment of Suspect Developmental Toxicants, p. 46324.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH

NIOSH is the research agency created by the OSH Act of 1970. NIOSH is a part of the CDC, which is a part of the Public Health Service which, in turn, is a part of DHHS. The director of NIOSH is appointed by the Secretary of HHS for a term of 6 years. NIOSH has no authority for promulgating or enforcing standards (risk management) but is responsible for conducting research and making recommendations to the Department of Labor pursuant to the OSH Act and the Federal Mine Safety and Health Act.

NIOSH research may begin at the urging of the Secretary of HHS, or on the initiative of the Director of NIOSH. An employer or employee request may also lead to a safety and health evaluation. In all its activities, NIOSH approaches the development and evaluation of standards with the intent of providing optimum protection for employees, whereas OSHA's mandate is to examine the potential costs and benefits (see chapter 7).

NIOSH has responsibility for several major activities:

1. develop criteria for recommended occupational safety and health standards,
2. conduct educational programs to provide an adequate supply of qualified personnel,
3. conduct informational programs on the importance of the use of adequate safety and health equipment,
4. conduct Health Hazard Evaluations, and
5. conduct industrywide studies of the effects of chronic or low-level exposures.

NIOSH has been criticized from several directions. OSHA has criticized it for the inadequacy of criteria documents for OSHA standard-setting. The General Accounting Office has criticized the quality of its criteria documents and Health Hazard Evaluation program. Labor groups have stated that it is unresponsive to worker requests. Management representatives have claimed that Health Hazard Evaluations are too aggressively pursued, and NIOSH research is of poor quality (for further discussion, see (29)). Recent directors of NIOSH have worked to improve the quality of NIOSH research.

Reproductive Health Hazard Research

Former and current NIOSH officials agree that NIOSH has been slow to study reproductive health hazards. This has been due, in part, to budgetary and personnel problems. In the last few years the issue of reproductive health hazard research has received higher priority (30). Current research activities are listed in table 6-6.

NIOSH has pursued several approaches for studying the adverse effect of occupation on human reproductive systems. First, NIOSH has accessed several large data bases that include information on occupation and has linked these data with State or city vital statistic and birth records, permitting an analysis that attempts to determine whether adverse pregnancy outcomes are associated with specific types of occupations. Second, NIOSH has been investigating the effects of specific exposure on both female and male reproductive function.

To study the effects on the female reproductive system, information on pregnancy outcomes from State or city records or information on pregnancy outcomes from a questionnaire administered to the mother is obtained and analyzed to determine if specific occupational exposures are associated with adverse pregnancy outcomes such as miscarriage, low birthweight, or malformations.

To study the effects on the male reproductive system, one of two strategies has been used: 1) a similar approach to the one described for study of effects following female exposure, except that the analysis determines whether adverse pregnancy outcomes of spouses are associated with specific occupational exposures of males; and 2) an evaluation of specific semen quality parameters. The parameters considered include sperm count, sperm motility, sperm morphology, and specific hormone activity. The meaning of these semen quality parameters in terms of actual adverse pregnancy outcomes is not known at present, but the study of these parameters is believed to document the effects of specific exposures.

Table 6-6.—NIOSH Reproductive Health Hazards Research

Subject of study/ suspected hazard	Status of research/ workers studied	As of Aug. 1, 1984
1. Oryzalin	Males	Completed
2. Carbon disulfide	Males and male workers' wives	Completed
3. Organic compounds (wastewater treatment workers)	Males	Completed
4. PCBs	Females	Completed
5. heavy metals (uranium workers)	Male workers' wives	Completed
6. DBCP	Males	Completed
7. Pharmaceutical estrogen	Males	Completed
8. Pharmaceutical lab workers	Females	Completed
9. EDB (2 studies)	Males	1 completed 1 in progress
10. Lead	Males	Nearly completed
11. Chemotherapeutic drugs	Females	1 study completed, hazard alert in preparation
12. Glycol ethers	Males	Field work completed, analysis in progress
13. Human semen characteristics	Male	Proposed
14. VDTs	Females	In progress
15. Dioxin	Males	Development stage
16. Ethylene oxide	Males and females	Proposed
17. Organo-tin compounds	Males	Interest
18. Butadiene	Males	Interest
19. Radiofrequency	Females	Abandoned (problem with cohorts) (but being reactivated)

NOTE: This list excludes some reports of health hazard evaluations based on clusters of negative reproductive outcomes (e.g., spontaneous abortions).

SOURCE: Office of Technology Assessment.

With respect to developmental toxicology, NIOSH has been conducting research on the effects of chemicals on the offspring of laboratory animals (rats) exposed during gestation. The tests used to determine the developmental effects examine both instinctive and spontaneous behavior. Using these study designs, NIOSH has studied several glycol ethers and industrial alcohols. The findings have shown that behavioral effects in the offspring can appear in the absence of other signs of toxicity in both the dam and the offspring.

NIOSH has a collaborative effort with the National Toxicology Program to test dose-response characteristics of selected chemicals for reproductive toxicity (30).

Reproductive Risk Assessment

Since NIOSH is a scientific and technical research agency, it approaches health hazard control with the view of providing maximum protection for workers. Thus, although it does not determine whether a risk is "significant" in the legal sense, it does attempt to quantify the magnitude of risk. Because the courts are requiring that OSHA standards contain increasingly detailed risk assessment, NIOSH has just initiated a formal section for quantitative risk assessment in the criteria documents division. Because the agency currently has little expertise in this field, it is working with consultants to develop the capability to better quantify the need for standards. One of the goals of the new section is to develop working groups in various subject areas and, where needed, to use outside experts to assist with risk assessments.

Exposure Estimates

NIOSH is in the process of surveying industries in order to estimate the numbers of individuals exposed to hazards. In contrast to an earlier survey, this is a representative sample of establishments selected from Dun & Bradstreet files. Supplementary samples of establishments from other files have been selected for the Standard Industrial Classifications determined to be inadequately covered by Dun & Bradstreet. The sample of establishments will constitute an unbiased random sample of industries in the United States. The sample design is based on a decision to maximize the reliability of estimates of numbers of employees exposed to hazards. Estimates by industry or estimates of the number of firms with hazards have been assigned lower priority. Information will be available by sex but not by age. Some data and tabulations are expected to be available by late 1985 (9,25).

Conclusions

Although NIOSH is carrying out a fair amount of research on reproductive health hazards, it lags behind the efforts of EPA in the development of reproductive and developmental risk guidelines. It is increasing this latter capability in response to court challenges of OSHA standards.

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Chapter 7

The Regulatory Process

CONTENTS

Page
Introduction 181

Occupational Safety and Health Administration and Related Agencies 181
  OSHA 181
  NIOSH 181
  OSHRC 182
  Exemptions from OSHA Jurisdiction due to Jurisdiction of Another Agency 182
  Congressional Appropriations Limitations 184
  OSHA's Authority to Regulate the Employment Relationship Due to Reproductive Hazards 185
  Employer and Employee Duties 187
  Procedures for Promulgation of Standards 192
  Hazard Identification 197
  Strategy for Hazard Exposure Control 198
  OSHA Reproductive Health Hazard Regulations 199
  Conclusions 208

EPA Authority to Address Reproductive Health Hazards 209
  Introduction: General Statutory Overview 209
  Toxic Substances Control Act 210
  Federal Insecticide, Fungicide, and Rodenticide Act 213
  EPA Implementation of Reproductive Health Hazard Control Programs 216
  Interagency Jurisdictional Issues 219
  Conclusions 221

Nuclear Regulatory Commission (NRC) 223
  Other Regulatory Authority 225
  NRC Regulations 225
  Temporary Workers 228
  Conclusions 230

List of Tables

Table No. *Page*
7-1. Judicial Review of OSHA Emergency Temporary Standards 196
7-2. States With Right-to-Know Laws 206
7-3. EPA Actions Under TSCA and FIFRA Based on Mutagenicity, Developmental, and Reproductive Effects 219

Chapter 7

The Regulatory Process

INTRODUCTION

Several Federal agencies have regulated substances based on deleterious health effects that include reproductive harm. While the Occupational Safety and Health Administration is the primary regulator of hazardous occupational exposures, occupational health issues are addressed by several other agencies as well. Each of these other agencies regulates industrial hazards in an area defined by either occupational category (e.g., the Mine Safety and Health Administration for mine workers) or type of exposure (e.g., the Environmental Protection Agency for pesticides).

This chapter addresses the issue of Federal Government regulation of workplace exposure to known and suspected reproductive health hazards. The activities of relevant Federal agencies are discussed, especially those of the Occupational Safety and Health Administration, the Environmental Protection Agency, and the Nuclear Regulatory Commission.

OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION AND RELATED AGENCIES

Prior to 1970, occupational safety and health regulation was nonexistent in a majority of States and consisted of a patchwork of sometimes inconsistent laws in the rest.[1] Congress, concerned with the human and economic costs of occupational injuries and illnesses, enacted the Occupational Safety and Health Act of 1970 (OSH Act) to "assure so far as possible every working man and woman in the Nation safe and healthful working conditions and to preserve our human resources."[2] Passage of national legislation concerned with workplace hazards brought occupational safety and health coverage to more than 75 million working Americans.[3] The OSH Act resulted in the creation of three agencies to deal with occupational safety and health issues on a national level: the Occupational Safety and Health Administration (OSHA), the National Institute for Occupational Safety and Health (NIOSH), and the Occupational Safety and Health Review Commission (OSHRC).

OSHA

OSHA is a regulatory agency within the Department of Labor. It sets mandatory health and safety standards, inspects workplaces to ensure compliance with those standards, and proposes penalties and abatement plans for employers found to be violating health and safety standards. OSHA also monitors the performance of State agencies operating State occupational safety and health plans under the OSH Act. In addition, OSHA provides education and consultation services to the public, workers, and employers, mostly through grant activities. OSHA is headed by a presidentially appointed Assistant Secretary of Labor for Occupational Safety and Health, to whom the Secretary of Labor has delegated authority under the OSH Act.

NIOSH

NIOSH conducts research and related activities leading to the development of criteria or recommendations for OSHA's use in setting health and safety standards. These activities include research designed to identify and evaluate workplace hazards, research concerning measurement techniques and control technologies, and education of health and safety professionals. NIOSH is part of the Centers for Disease Control (CDC) of the U.S. Public Health Service (PHS), which is within the Department of Health and Human Services (DHHS). NIOSH is headed by a Director appointed by the Secretary of HHS for a term of 6 years.

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[1] U.S. Congress, Office of Technology Assessment, Preventing Illness and Injury in the Workplace (1985).

[2] 29 U.S.C. § 651ff.

[3] See generally U.S. Congress, Office of Technology Assessment, Preventing Illness and Injury in the Workplace (1985).

The separation of research and regulatory standard-setting into NIOSH and OSHA is controversial. While defended by some as a way of keeping scientific activities neutral, it has also been said to lead to inefficiency and duplication, and the activities of the two agencies have been criticized as insufficiently coordinated.[4] (See box 7A.)

OSHRC

OSHRC is an independent, quasi-judicial review board whose duties are limited to reviewing OSHA citations issued to employers charged with violating OSHA standards. In deciding these cases, however, OSHRC decides the nature and scope of many employer obligations concerning employee health and safety. OSHRC is composed of three members, appointed by the President with the advice and consent of the Senate, for staggered terms of 6 years.

Exemptions From OSHA Jurisdiction Due to Jurisdiction of Another Agency

Most workers are covered by the OSH Act. (A detailed description of covered employers and employees appears in a staff paper available from OTA.) Section 4(b)(1) of the Act provides that the statute does not apply to:

> . . . working conditions of employees with respect to which other Federal agencies . . . exercise statutory authority to prescribe or enforce standards or regulations affecting occupational safety and health.

Although Congress intended to avoid duplication or conflict among Federal agencies that regulate safety and health, there have been many questions as to which working conditions are exempt from application of the OSH Act, what the limits of exemptions are, and what the procedural implications of exemptions are. (The legal principles governing exemption from OSHA jurisdiction are discussed in detail in a staff paper available from OTA.)

Recent Commission decisions suggest a three-part test to determine whether OSHA is preempted from exercising jurisdiction by virtue of § 4(b)(1):

1. The working condition is covered by another Federal act exclusively directed at employee safety and health or more generally directed at public safety and health, and employees directly receive the protection the act is intended to provide.
2. The other Federal agency has exercised its statutory grant of authority.
3. The other Federal agency has acted in such a manner as to exempt the cited working conditions from OSHA jurisdiction.

Relevance to Reproductive Health Hazards

There are two principal ways in which the issue of § 4(b)(1) preemption may be relevant to OSHA's regulation of reproductive health hazards. The first involves OSHA's attempt to promulgate standards covering working conditions regulated by another Federal agency. For example, in 1973, OSHA issued an emergency temporary standard (ETS) for exposure to 21 organophosphorous pesticides.[5] The standard required employers to warn employees of pesticide hazards, set field reentry times, and prescribed sanitation and medical services and first aid. In 1974, the Fifth Circuit stayed and then vacated the ETS on the ground that no "grave danger" existed, as required by § 6(c).[6]

After the Fifth Circuit's decision, OSHA held hearings on a new permanent pesticide standard. Eventually, OSHA discontinued its rulemaking and acceded to the position of the Environmental Protection Agency (EPA) that OSHA was preempted from regulating pesticides because of EPA's authority under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA).[7] In a subsequent law-

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[4] N. Ashford, Crisis in the Workplace: Occupational Disease and Injury (1976).

[5] 38 Fed. Reg. 10,715 (1973).

[6] Florida Peach Growers Association v. U.S. Department of Labor, 489 F.2d 120 (5th Cir. 1974).

[7] See B. Mintz, OSHA: History, Law and Policy 105 (1984).

Box 7A.—Interagency Relations

In researching the status of relations between OSHA and NIOSH, which are integral to the rule-making process for reproductive and other health hazards, a number of interviews were conducted with present and former OSHA and NIOSH officials.

Certain patterns emerged from their responses. Present officials tended to be positive about interagency relations. Former officials were largely negative about both past and present relations. High-ranking officials were more positive about interagency relations than were their subordinates.

The interviews focused on four main subject areas: institutional concerns, funding and personnel, priorities and policies, and interagency programs.

Institutional Concerns.—Perceptions of the missions of NIOSH and OSHA differ. A close working relationship between the assistant secretary of labor for OSHA and the NIOSH director during the Carter Administration was criticized for ostensibly jeopardizing the agency's image as a neutral research body.[8] The former NIOSH director, while upholding the scientific accuracy of NIOSH research, responded that the goal of both agencies is to protect workers, and that "the law never says that NIOSH has to be neutral."

Reagan Administration officials favor the clear separation of research and regulation. A former assistant secretary of labor for OSHA in the Reagan Administration contends that NIOSH's role is, and should be, limited to research, a view shared by the current NIOSH director. NIOSH and OSHA have consequently discontinued the practice of publishing joint statements and hazard alerts, which had been seen as having greater impact on the public due to having been issued by both agencies.

Interaction between the agencies may be hampered by their differing levels in the bureaucracy, according to a former NIOSH director. OSHA's head functions directly under the Secretary of Labor, whereas the director of NIOSH is responsible to the director of CDC, who is responsible to the Assistant Secretary for Health, who reports in turn to the Secretary of HHS.

Other officials disagreed, and the current NIOSH director suggested that NIOSH's "insulation" may be advantageous in that it frees the Institute's director to work exclusively on science while other officials tend to the regulatory burdens.

A former OSHA chief and a NIOSH director who served under both Democratic and Republican administrations expressed concern that funding for complementary programs can be jeopardized when NIOSH and OSHA budget requests are reviewed by different budget examiners at the Office of Management and Budget (OMB). The current NIOSH director does not consider this to be a problem, however.

Funding and Personnel.—It is widely agreed that personalities have an important effect on the interagency relationship. Exchanges of personnel and other joint programs can improve the relationship, a Carter Administration OSHA head believes, but opponents tend to view such efforts as "entanglement."

The most common criticism of current OSHA-NIOSH relations is that reductions in technical personnel at OSHA limit the agency's capacity for in-depth review of NIOSH's work. (OSHA's Directorate of Health Standards Programs has only one toxicologist, two epidemiologists, and no physicians, although the Directorate of Technical Support has additional personnel).[9] OSHA's present lack of technical expertise, according to a NIOSH official, renders OSHA-NIOSH relations "close to nonexistent at the working level."

An OSHA official agrees that chronic personnel shortages impair the agency's ability to perform technical reviews. The only full-time occupational physician at OSHA, he has been aided by in-house physicians on interagency assignments, by four residents (who serve 2- to 4-month residencies), and by expert consultants when needed. However, the residency program may be in jeopardy. A senior OSHA official, who acknowledges that NIOSH generates more technical material than OSHA can handle, doubts that more technical staff is the answer. In his view, more lawyers, more administrators, and more staff are required all the way up the line.[10]

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[8] See U.S. Congress, Office of Technology Assessment, Preventing Illness and Injury in the Workplace (1985).

[9] According to the Administrative Officer of OSHA's Directory of Health Standards, as of Aug. 1, 1984, OSHA had 25 professionals in the Health Standards Directory (includes health scientists and industrial hygienists), compared with a high of 40 in March 1981. There are presently two epidemiologists and one toxicologist; this compares with the 1979 high of five to six epidemiologists and one toxicologist.

[10] The decline in scientific and nonscientific personnel at OSHA between March 1979 and October 1983 is documented in a recent report of the General Accounting Office. 14 O.S.H. Rep. (BNA) 281 (1984).

Priorities and Policies.—Officials of each agency indicate disapproval of the priorities and policies of the other. NIOSH officials cite difficulties in getting OSHA feedback on NIOSH-proposed criteria documents needed to enable NIOSH to set research priorities, frustration at OSHA's failure to implement NIOSH's scientific recommendations, and the need for OSHA to reinstitute a policy of specifying its scientific requirements. An earlier lack of CDC support for NIOSH goals, cited by former NIOSH officials, is believed by the current NIOSH director to have been ameliorated by NIOSH's move from Rockville, Maryland, to Atlanta, Georgia, which is CDC's location.

OSHA officials claim that NIOSH research does not always adapt to OSHA's regulatory goals, and in some instances is of questionable quality.

Interagency Programs.—The general framework for OSHA-NIOSH cooperative programs is set out in a 1979 interagency agreement.[11] In broad terms, the agreement establishes each agency's responsibilities in development of health and safety criteria, development and revision of health and safety standards, health hazard evaluations, compliance assistance, technical information exchange, and other matters. Although the agreement is believed to provide a good framework, it has not been followed; none of the current OSHA officials interviewed for this assessment were even aware of its existence. Most earlier interagency programs are no longer in effect, though NIOSH and OSHA officials continue to meet regularly.

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[11] 44 Fed. Reg. 22,834 (1979).

suit brought by a farmworker group to compel OSHA to issue a pesticide standard, the D.C. Circuit held that OSHA was indeed preempted under § 4(b)(1) by virtue of the Federal Environmental Pesticide Control Act (FEPCA) (which revised FIFRA).[12] Thus, OSHA was not permitted to issue a standard for a class of hazards that EPA was authorized to regulate.

The second way in which § 4(b)(1) may be relevant to OSHA's regulation of reproductive health hazards involves attempts by OSHA to prohibit allegedly discriminatory reproductive health policies of employers. In *American Cyanamid,*[13] a case discussed more fully later in this chapter, the employer was cited under § 5(a)(1) (the general duty clause, discussed below) after five women employed in the lead pigments department submitted to surgical sterilization in order to retain their jobs. In granting the employer's motion for a judgment in its favor, the Commission administrative law judge (ALJ) held, among other things, that § 4(b)(1) precludes OSHA from exercising authority because the employer's fetal protection policy is possibly an unfair labor practice under the National Labor Relations Act and possibly sex discrimination under Title VII of the Civil Rights Act of 1964. Although the Commission subsequently affirmed the ALJ's decision on other grounds, the plain language of § 4(b)(1) would seem to preclude the ALJ's interpretation. Neither the National Labor Relations Board nor the Equal Employment Opportunity Commission are agencies which "exercise statutory authority to prescribe or enforce standards or regulations affecting occupational safety or health." The Commission's decision was affirmed on other grounds by the U.S. Court of Appeals for the District of Columbia.

Congressional Appropriations Limitations

Beginning with fiscal 1977, Congress has restricted some specific aspects of OSHA enforcement by attaching limitations to OSHA appropriations bills and continuing resolutions. Five of these limitations are relevant to OSHA regulation of reproductive health hazards in the workplace.

First and most importantly, OSHA is prohibited from inspecting workplaces with 10 or fewer employees in industries with three-digit Standard Industrial Classification (SIC) injury and illness rates below the national lost workday injury rate for manufacturing (currently 3.4 per 100 employees).[14] The SIC codes and the injury rate are both determined by the Bureau of Labor Statistics. The injury rate is updated annually. There are several exceptions to the limitation, and inspections are still permitted in the following instances: in response to complaints, for failure to correct, for willful violations, to investigate accidents, for imminent dangers, for health hazards, and to investigate discrimination complaints.

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[12] Organized Migrants in Commun. Action, Inc. v. Brennan, 520 F.2d 1161 (D.C. Cir. 1975). See Comment, Interpreting OSHA's Preemption Clause: Farmworkers As A Case Study, 128 U. Pa. L. Rev. 1509 (1980).

[13] American Cyanamid Co., 9 O.S.H. Cas. (BNA) 1596 (1981) aff'd, Oil, Chemical & Atomic Workers International Union v. American Cyanamid Co., 741 F.2d 444 (D.C. Cir. 1984). A second case against Cyanamid, brought by female employees, is discussed in ch. 8.

[14] See OSHA Instruction 2.51B (1984).

Second, OSHA is prohibited from inspecting workplaces for 6 months after a State inspection is performed in States with approved plans, except for investigation of employee complaints and fatalities, special studies, and accompanied monitoring visits.

Third, OSHA is prohibited from assessing penalities for first-instance nonserious violations of any employer unless the inspection discloses 10 or more violations. OSHA is still permitted to issue citations that prescribe an abatement date for these violations, and second-instance violations of any nature can carry a penalty.

Fourth, farms, ranches, orchards, and related operations with 10 or fewer employees at one time during the past year, except those with migrant labor camps, are exempt. Members of a farm employer's immediate family are not considered employees.

Finally, no penalties may be assessed against an employer with 10 or fewer employees who had a prior onsite consultation and had made good faith efforts to abate the violative conditions prior to the inspection.

OSHA's Authority to Regulate the Employment Relationship Due to Reproductive Hazards

Medical Removal Protection and Rate Retention

One possible way of addressing the problem of reproductive health hazards in the workplace is for OSHA to regulate the permissible range of an employer's options relating to employee exposure. For example, OSHA might promulgate a standard prohibiting an employer from excluding only women (or men) from areas where there is exposure to known or suspected reproductive or developmental hazards; that is, abortifacient, mutagenic, teratogenic, or embryo-fetotoxic substances. The promulgation of such a regulation would raise the legal issue of whether OSHA had exceeded its statutory authority.

Although the courts have not addressed the issue of OSHA's authority to promulgate a standard prohibiting exclusionary employment practices, some analogous issues have arisen in cases involving medical removal protection (MRP) and rate retention (RR). MRP is simply the removal of employees from further hazardous exposure to a toxic substance until it is medically advisable to return. RR requires that the removed employee's wages and benefits be maintained during the period of removal.

MRP and RR provisions in OSHA health standards have become increasingly stringent. For example, the vinyl chloride standard (promulgated in 1974) provides for MRP, but not RR.[15] The asbestos standard (promulgated in 1972) provides for MRP for employees for whom respirators are ineffective, but RR is required only if there is an available position.[16] The cotton dust standard (promulgated in 1978), however, squarely raised the issue of OSHA authority by requiring RR for certain employees.[17] The Supreme Court, without deciding the issue of whether OSHA could impose MRP and RR requirements at all, struck down this RR provision because OSHA "failed to make the necessary determination or statement of reasons that its wage guarantee requirement is related to the achievement of a safe and healthful work environment."[18]

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[15] 29 CFR § 1910.1017(k)(5) (1984).

[16] Ibid. at § 1910.1001(d)(2)(iv)(c).

[17] The cotton dust standard, 29 CFR § 1910.1043 (1984), allowed reliance on the use of respirators to protect employees from exposure to cotton dust during the 4-year interim period given employers to install engineering controls. (After 4 years, respirators were not allowed except in limited cases.) One part of the respirator provision required employers to give employees unable to wear a respirator (because of facial irritation, severe discomfort, or impaired breathing) the opportunity to transfer to another position, if available, where the dust level meets the standard's permissible exposure limit (PEL). When such a transfer occurs the employer must guarantee that the employee's wages and benefits are maintained.

[18] American Textile Manufacturer's Institute, Inc. v. Donovan, 452 U.S. 490, 537-38 (1981). Rather than explaining the RR provision as being essential in ensuring that workers would seek needed MRP, OSHA had stated that the "goal of this provision is to minimize any adverse economic impact on the employee by virtue of the inability

The Court's most instructive statement on the permissible scope of OSHA rulemaking is the following:

> Because the Act in no way authorizes OSHA to repair general unfairness to employees that is unrelated to achievement of health and safety goals, we conclude that OSHA acted beyond statutory authority when it issued the wage guarantee regulation.[19]

When OSHA subsequently promulgated its revised lead standard in 1978, it included an even broader MRP and RR provision.[20] When an employee is removed in any way, the employee retains his or her earnings rate, seniority, and benefit levels for up to 18 months and on return must be restored to his or her original job status.

Unlike its statement of reasons accompanying the cotton dust standard, the lead standard contained detailed findings of the need for RR. OSHA found that "unless workers were guaranteed all their wage and seniority rights on removal, they would resist cooperating with the medical surveillance program that determined the need for removal, since they reasonably might fear being fired or sent to lower paying jobs if they revealed dangerously high blood-lead levels."[21] This rationale was upheld by the D.C. Circuit.[22]

The D.C. Circuit's opinion contains a footnote with particular relevance to the issue of MRP and reproductive health hazards:

> Amici representing public interest law organizations and California State labor agencies have argued that MRP is not only legally valid under the OSH Act, but is legally required by Title VII of the Civil Rights Act of 1964, 42 U.S.C. § 2000(e) et seq. (1976 & Supp. II 1978). They argue that without MRP employers will discriminate against fertile women—to whom lead exposure poses an even greater threat than it does to other workers—by excluding them from all lead-exposed jobs at the outset.
>
> A review of an OSHA proceeding, however, is not the place to address hypothetical Title VII questions, and in any event we think fertile women can find statutory protection from such discrimination in the OSH Act's own requirement that OSHA standards ensure that "*no employee* will suffer material impairment of health." 29 U.S.C. § 655(b)(5) (1976) (emphasis added).[23]

The cotton dust and lead cases suggest that OSHA may promulgate health standards that provide for medical removal and rate retention, so long as any rate retention requirement is related to the achievement of a healthful work environment, rather than to redress unfairness or discrimination.

When read together, the cotton dust and lead cases suggest the following about OSHA regulation of reproductive health hazards:

1. OSHA has the statutory authority to protect the sexual and reproductive health of male and female workers. The reproductive functions of these workers include the ability to produce healthy offspring. OSHA therefore has apparent authority to protect embryos/fetuses from workplace hazards.

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to wear a respirator." Id. at 538 (quoting 43 Fed. Reg. 27,387 (1978)). The Court dismissed OSHA's statement of the importance of encouraging employees to disclose symptoms of disease as unacceptable "post-hoc rationalizations." Id. at 539.

[19] 452 U.S. at 540 (footnote omitted).

[20] The current version of OSHA's lead standard is at 29 CFR § 1910.1025 (1984).

[21] 43 Fed. Reg. 54,442-46 (1978).

[22] United Steelworkers of America v. Marshall, 647 F.2d 1189 (D.C. Cir. 1980), cert. denied sub nom. Lead Indus. Assn. Inc. v. Donovan, 453 U.S. 913 (1981). In this case, the D.C. Circuit upheld the validity of the MRP and RR provision. The lead industry had argued that Congress did not intend to have MRP and RR under OSHA because the Act is silent on this subject, while the Coal Mine Health and Safety Act of 1969 (CMHS Act), passed the year before OSHA, contained an MRP provision. The court rejected this argument, noting that the CMHS Act covered a single industry and was drafted with much greater specificity than OSH Act. The lead industry also argued that the provision violated § 4(b)(4)'s prohibition on OSHA interfering with workers' compensation. Although acknowledging the "seriousness" of this argument, the court noted the limited duration and scope (e.g., there is no payment for medical expenses) of RR benefits, and indicated that the group of workers to benefit from this provision will become increasingly smaller as the PEL is lowered. "We conclude that though MRP may indeed have a great practical effect on workmen's compensation claims, it leaves the state schemes wholly intact as a legal matter, and so does not violate § 4(b)(4)." 647 F.2d at 1236. Finally, the court rejected the argument that MRP and RR violates the national labor policy of allowing all substantive provisions of labor management relations to be left to collective bargaining. Simply because earnings protection is a mandatory subject of bargaining and could be adopted through collective bargaining does not mean OSHA has no authority to mandate such a program. The Supreme Court refused to hear the case, thereby allowing the D.C. Circuit's decision to stand.

[23] 647 F.2d at 1238 n.74 (emphasis in original).

2. OSHA could promulgate a single permissible exposure level that protects male workers, female workers, and embryos/fetuses from a hazardous substance, so long as the standard met all of the requirements of §§ 3(8) and 6(b)(5), such as "significant risk" and technological and economic feasibility.
3. OSHA might be precluded from promulgating a regulation directed only at prohibiting the exclusion of all women from exposure to reproductive health hazards. Such rulemaking could be held to be preempted by Title VII, pursuant to § 4(b)(1), or might be considered to be an *ultra vires* attempt "to repair general unfairness unrelated to achievement of health and safety goals," as held in the cotton dust case. However, OSHA can allow the exclusion of men and women under a specific standard addressing health and safety goals (e.g., lead standard).
4. OSHA could probably enact a regulation prohibiting an employer from making sterilization of current employees (male, female, or all employees) a condition of continued employment. Although the *American Cyanamid* case (see note 13) held that the general duty clause does not implicitly prohibit such employer practices, an explicit regulation might do so. Valid health and safety goals would seem to include prohibiting both exposure to sterilizing agents and "voluntary" sterilization in order to retain employment. Note that an employment policy requiring that all employees be sterilized would not violate Title VII because both sexes are treated equally. It is less clear whether OSHA has the authority to promulgate a regulation prohibiting an employer from hiring only employees who had been sterilized or were otherwise incapable of reproduction. Such a regulation might be upheld based on the same considerations as are applicable to current employees.
5. The promulgation of an OSHA standard prohibiting an employer from refusing to hire fertile women would entail elements of both considerations 3 and 4. The legality of such rulemaking may ultimately turn on the state of the factual record developed at the rulemaking, including evidence as to whether prohibiting the employment of fertile women causes women to become sterilized.[24]

Employer and Employee Duties

OSHA imposes duties on both employers and employees. Employers are required: 1) to comply with OSHA standards, and 2) to generally provide employment free from recognized hazards. Employees are also required to comply with OSHA standards, though final responsibility for employee compliance rests with the employer. These duties are discussed below.

The OSH Act is enforced solely by the Federal Government and, in States with approved plans, by those States. Specifically, OSHA inspects workplaces for compliance with OSHA standards and workplace, and may issue citations to noncompliant employers. There is no private right of action to enable employees to obtain enforcement of OSHA standards or the general duty clause as to their employers.

Compliance With Standards—Section 5(a)(2)

Section 5(a)(2) of the Act provides simply that each employer "shall comply with occupational safety and health standards promulgated under this Act." Whether an employer has complied with the Act is not determined by the number of accidents that have taken place. Furthermore, the occurrence of an accident does not always mean there has been a violation.[25] Even the occurrence of hazardous conduct is not per se evidence of a violation.[26] Conversely, the absence of an accident does not mean there was no violation—it may only reflect the employer's good fortune.[27] Even a serious violation does not require any actual death or physical injury.[28] The Act seeks to prevent injury and illness by eliminating hazardous conditions.

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[24] M. Rothstein, The Regulation of Reproductive Hazards Under OSHA (Aug. 1984) (unpublished report).

[25] B & B Insulation, Inc. v. OSHRC, 583 F.2d 1364, 1372 & n.17 (5th Cir. 1978); Ryder Truck Lines, Inc. v. Brennan, 497 F.2d 230, 233 (5th Cir. 1974); Lebanon Lumber Co., 1 O.S.H. Cas. (BNA) 1165 (1973).

[26] National Realty & Construction Co. v. OSHRC, 489 F.2d 1257, 1266 (D.C. Cir. 1973).

[27] A.E. Burgess Leather Co. v. OSHRC, 576 F.2d 948, 951 (1st Cir. 1978); Arkansas-Best Freight System, Inc. v. OSHRC, 529 F.2d 649, 655 (8th Cir. 1976); General Electric Co., 7 O.S.H. Cas. (BNA) 2183 (1980); Kroehler Manufacturing Co., 6 O.S.H. Cas. (BNA) 2045 (1978).

Environmental Monitoring.—The employer is responsible for conducting periodic atmospheric tests to determine the presence and concentration of hazardous substances that are addressed by OSHA standards. The standards differ on the frequency of the testing, but even the most stringent requirements have been upheld.

OSHA's health standards often rely on the concept of an *action level*. For example, in the ethylene oxide standard, OSHA established a one part per million (ppm) 8-hour time-weighted average (TWA) as the exposure limit. The action level was set at 0.5 ppm. When initial monitoring reveals exposures below the action level, no further monitoring is required unless there is a change in production, process, or control. If exposures are above the action level, exposures must be monitored twice per year. Monitoring may be discontinued, however, if two consecutive measurements, taken at least 7 days apart, show exposures below the action level. If exposures go above the TWA, more frequent monitoring is required as well as reductions in exposure levels. These requirements are summarized in the following table:

Exposure scenario	*Required monitoring activity*
Below the action level	No monitoring required
At or above the action level, but at or below the TWA	Monitoring exposures two times per year
Above the TWA	Monitor exposures four times per year

The action level attempts to provide a margin of safety, so that it is unlikely that a minor fluctuation in atmospheric concentration would result in an exposure exceeding the TWA. It requires that employers with exposures approaching the TWA keep close measurements to ensure that the TWA is not exceeded, while removing the burden of continuous environmental monitoring from employers with only slight exposure levels. The main problem with the use of the action level concept is that it eliminates important protections for workers whose exposures are below the action level. For example, in the *Benzene* case, the Supreme Court was critical of OSHA for not requiring monitoring and medical testing of employees exposed below the action level:

> By doing so, [OSHA] could keep a constant check on the validity of the assumptions made in developing the permissible exposure limit, giving it a sound evidentiary basis for decreasing the limit if it was initially set too high. Moreover, in this way it could ensure that workers who were unusually susceptible to benzene could be removed from exposure before they had suffered any permanent damage.[29]

A similar problem exists under the lead standard, which established a permissible exposure limit (PEL) of 50 micrograms of lead per cubic meter of air averaged over an 8-hour work day and an action level of 30 micrograms. An employer's duty to supply protective clothing, change rooms, showers, and other hygiene facilities and practices is contingent on the exposure level being above the action level.[30] However, an action level that is sufficient to protect the worker may not be sufficient to protect a child exposed to the worker's contaminated clothing.

Biological Monitoring.—OSHA health standards may require the biological monitoring of exposed employees to measure the body's uptake of toxic substances. For example, the lead standard requires that the employer provide blood sampling and analysis for lead and zinc protoporphyrin levels for each employee with lead exposure at or above the action level. This monitoring is required at least every 6 months.[31]

Medical Surveillance.—OSHA's 22 health standards regulating toxic substances require a variety of medical procedures. In general, employers must conduct preplacement examinations, a physician must furnish employers with a statement of suitability for employment in the regulated area, the employer must conduct periodic (usually annual) examinations, and in some instances the employer must conduct examinations at termination of employment. The failure to conduct these required medical examinations may lead to the issuance of OSHA citations and the assessment of penalties.

OSHA medical surveillance programs have two primary purposes: 1) to give the employee notice of any adverse health effects that he or she may have suffered so that proper medical attention may be obtained and precautionary measures taken, and 2) to provide OSHA and NIOSH with data for research purposes.[32] (The mechanics of medical surveillance programs are discussed in Appendix C-1.)

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[28] Brennan v. Butler Lime & Cement Co., 520 F.2d 1011, 1017 (7th Cir. 1975).

[29] Industrial Union Department v. American Petroleum Institute, 448 U.S. 607, 658 (1980) (footnotes omitted).

[30] 29 CFR § 1910.1025(g), (i) (1984).

[31] Id. at § 1910.1025(j)(2).

Controls/Other Requirements.—OSHA health standards attempt to reduce exposure through a variety of control strategies, such as engineering controls, work practice controls, personal protective equipment, and administrative controls.[33]

The General Duty Clause—Section 5(a)(1)

Section 5(a)(1) of the Act, the general duty clause, provides that each employer "shall furnish to each of his employees employment and a place of employment which are free from recognized hazards that are causing or are likely to cause death or serious physical harm to his employees."

The general duty clause was enacted to cover serious hazards to which no specific standard applies. Because the general duty clause was designed to augment rather than supplant standards, citation under § 5(a)(1) is improper where a specific standard is appropriate.[34] During the first few years of the Act's existence, the general duty clause was used to prohibit hazardous conduct while specific standards were being promulgated or before a standard's effective date.[35] Subsequently, however, the general duty clause has been used for peculiar violations not covered by specific standards.[36]

The most distinctive and significant element of general duty clause violations is that they are limited to "recognized hazards." The recognition requirement serves to ensure that cited employers have at least constructive knowledge of the existence of specific hazardous conditions. In this way, Congress sought to eliminate the unfairness of assessing first-instance civil penalties based on such a sweeping and broadly worded provision.[37]

A hazard is considered recognized: 1) if it is common knowledge in the employer's industry, or 2) if the employer had actual or constructive knowledge of the hazardous condition. Recognition thus may be established either objectively or subjectively.[38]

Industry Recognition of Hazard.—In addition to expert testimony, the Commission and courts have held that other sources may be used to prove industry recognition of a hazard. State[39] and local[40] laws, American National Standards Institute[41] and National Fire Protection Association[42] standards, industry publications,[43] and manufacturer's warnings[44] all have been used to demonstrate that a hazard was recognized by the employer's industry. It is essential that the referenced industry is the appropriate one.[45] All industries do not necessarily recognize the same hazards and a citation may be vacated on this basis.[46]

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[32] B. Mintz, OSHA: History, Law, and Policy 131 (1984).

[33] Depending on the working conditions, employers may have a wide range of other duties, such as providing showers and changing rooms, protective clothing, and laundry facilities. Employers also may be obligated to post warning signs and give detailed warnings to their employees. Finally, OSHA standards require that all health hazard emergencies be reported. For example, carcinogen exposure must be reported to OSHA within 24 hours. See, e.g., 29 CFR § 1910.1003(f)(2) (1984). Radiation exposure must be reported immediately by phone or telegram and a written report must be filed within 15 days. 29 CFR § 1910.96(1) (1984).

[34] Brisk Waterproofing Co., 1 O.S.H. Cas. (BNA) 1263 (1973). See S. Rep. No. 1282, 91st Cong., 2d sess. 9, 10, reprinted in 1970 U.S. Code Cong. & Ad. News 5177, 5185-86.

[35] See American Smelting & Refining Co. v. OSHRC, 501 F.2d 504, 512 (8th Cir. 1974).

[36] See, e.g., Marquette Cement Manufacturing Co., 3 O.S.H. Cas. (BNA) 1928 (1976), vacated, 568 F.2d 902 (2d Cir. 1977) (employee crushed by falling bricks); Richmond Block, Inc., 1 O.S.H. Cas. (BNA) 1505 (1974) (employee killed while cleaning inside of cement mixer); Southern Soya Corp., 1 O.S.H. Cas. (BNA) 1412 (1973) (employees suffocated by cave-in of stored cottonseed).

[37] The case of American Smelting & Refining Co. v. OSHRC, 501 F.2d 504 (8th Cir. 1974), concerned the issue of whether recognized hazards are limited to those detectable through the senses or whether they extend to hazards only detectable through instrumentation. The Eighth Circuit reviewed the legislative history of the general duty clause and found of considerable importance the fact that Congress changed the wording from "readily apparent hazards," used in an earlier version of the bill, to "recognized hazards." Moreover, the court pointed out that the ameliorative purpose of the Act would be subverted by a narrow construction of "recognized hazards." "[T]o limit the general duty clause to dangers only detectable by the human senses seems to us to be a folly. . . . Where hazards are recognized but not detectable by the senses, common sense and prudence demand that instrumentation be utilized." Id. at 511.

[38] In National Realty & Construction Co. v. OSHRC, 489 F.2d 1257 (D.C. Cir. 1973), a landmark § 5(a)(1) case, the D.C. Circuit held that whether a hazard is recognized by an industry is determined by the "common knowledge of safety experts who are familiar with the circumstances of the industry or the activity in question." Ibid. at 1265 n.32. The Commission has followed National Realty and also has held that the expert testimony of a compliance officer about industry practice may be used to show that a hazard was recognized. Beaird-Poulan, 7 O.S.H. Cas. (BNA) 1225 (1979); Cormier Well Service, 4 O.S.H. Cas. (BNA) 1085 (1976).

[39] Ford Motor Co., 5 O.S.H. Cas. (BNA) 1765 (1977); Sugar Cane Growers Coop., 4 O.S.H. Cas. (BNA) 1320 (1976); M.A. Swatek & Co., 1 O.S.H. Cas. (BNA) 1191 (1973).

[40] Williams Enterprises, Inc., 4 O.S.H. Cas. (BNA) 1663 (1976).

[41] St. Joe Minerals Corp. v. OSHRC, 647 F.2d 840, 845 n.8 (8th Cir. 1981); Betten Processing Corp., 2 O.S.H. Cas. (BNA) 1724 (1975).

[42] Cargill, Inc., 10 O.S.H. Cas. (BNA) 1398 (1982).

[43] R. L. Sanders Roofing Co., 7 O.S.H. Cas. (BNA) 1566 (1979), rev'd, 620 F.2d 97 (5th Cir. 1980).

[44] Young Sales Corp., 7 O.S.H. Cas. (BNA) 1297 (1979), aff'd. mem., No. 79-1612 (D.C. Cir. 1980).

Employer Knowledge of Hazard.—An employer's knowledge that a condition is hazardous does not depend on the occurrence of prior accidents.[47] Moreover, employer knowledge encompasses both actual and constructive knowledge. Thus, employer knowledge has been found on the basis of correspondence, industry meetings, and publicized accidents;[48] warnings given to supervisors by an independent engineering firm and at least one of its own employees;[49] the employer's use of fences, warning lights, and requiring passes to the area;[50] and the employer's taking some measures to protect exposed employees.[51]

Companies and industries thus have little incentive to participate in epidemiologic studies of workers exposed to possible occupational health hazards. Such studies can be used to establish the existence of a "recognized hazard," thereby creating for companies a legal duty to abate the hazard under the general duty clause. These studies can also be used to support tort liability (chapter 10) and workers' compensation (chapter 9) claims. Without industry cooperation, however, it is difficult for academic and government researchers to learn more about occupational health hazards.

In *National Realty,* the D.C. Circuit outlined the Secretary of Labor's burden of proving a violation of the employer's general duty. The Secretary must prove: 1) that the employer failed to render its workplace free of a hazard that was 2) recognized, and 3) causing or likely to cause death or serious physical harm, and 4) that the citation has specified the particular steps the cited employer should have taken to avoid citation and that these measures are feasible and have a likely utility.[52]

The General Duty Clause and Reproductive Health Hazards.—There are two possible ways in which § 5(a)(1) may be relevant to reproductive hazards in the workplace. First, employers could be issued citations under § 5(a)(1) and ordered to abate working conditions that are harmful to the reproductive health of workers or their offspring. The Secretary of Labor, however, would have two difficult hurdles to overcome in proving such a violation. To begin with, citation under § 5(a)(1) requires the hazard to be recog-

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[45] See R.L. Sanders Roofing Co. v. OSHRC, 620 F.2d 97 (5th Cir. 1980)(Commission erred in looking to construction industry rather than roofing industry).

[46] See, e.g., H-30, Inc. v. Marshall, 597 F.2d 234 (10th Cir. 1979).

[47] St. Joe Minerals Corp. v. OSHRC, 647 F.2d 840, 845 n.7 (8th Cir. 1981). Cf. Magma Copper Co. v. Marshall, 608 F.2d 373 (9th Cir. 1979) (where recognition is based on employer knowledge the Secretary has the burden of demonstrating that the employer's safety precautions were unacceptable in its industry).

[48] Atlantic Sugar Association., 4 O.S.H. Cas. (BNA) 1355 (1976).

[49] St. Joe Minerals Corp. v. OSHRC, 647 F.2d 840, 845 (8th Cir. 1981).

[50] General Electric Co., 10 O.S.H. Cas. (BNA) 2034 (1982).

[51] Wheeling-Pittsburgh Steel Corp., 10 O.S.H. Cas. (BNA) 1242 (1981). Some recent decisions of the Commission, Litton Systems, Inc., 10 O.S.H. Cas. (BNA) 1179 (1981), and courts of appeals, Donovan v. Missouri Farmers Association, 674 F.2d 690 (8th Cir. 1982); Continental Oil Co. v. OSHRC, 630 F.2d 446 (6th Cir. 1980), cert. denied, 450 U.S. 965 (1981), have inferred employer knowledge from the obvious nature of the hazard. For example, in one case, the Commission found an "obvious" hazard where the employer refueled gasoline-powered trucks indoors in the vicinity of open-flame heaters. Eddy's Bakeries Co., 9 O.S.H. Cas. (BNA) 2147 (1981).

[52] National Realty & Construction Co. v. OSHRC, 489 F.2d 1257 (D.C. Cir. 1973).

nized by the employer or its industry. **For newly discovered or suspected-but-unproven reproductive health hazards, it may be difficult to prove that they were actually or constructively recognized as hazardous. Thus, the general duty clause is unlikely to be a substitute for an emergency standard under § 6(c) as an interim measure until section § 6(b) rulemaking is completed.**

The other problem with using the general duty clause to cite employers for hazardous conditions is that the clause cannot be used unless there is no applicable standard under § 5(a)(2). For example, if a standard had a PEL of 10 ppm and the data showed that there were still reproductive health effects at exposures below the PEL, the general duty clause could not be used. The Commission has held that citation under § 5(a)(1) is improper where the applicable standard is inadequate, because this would amount to a circumvention of the rulemaking process.[53]

OSHA's enforcement guidelines[54] also provide that the general duty clause may not be used to require an abatement method not set forth in a specific standard. For example, if a standard provides for engineering controls but not medical surveillance, § 5(a)(1) may not be cited to require medical surveillance.

A second possible use of the general duty clause, to prohibit exclusionary employment practices, has already been attempted unsuccessfully. In *American Cyanamid Co.,* the only case to address this issue,[55] the Commission was faced with the question of whether the employer's policy, which excluded from certain employment women aged 16 to 50 who had not been surgically sterilized, constituted a "hazard" under § 5(a)(1). Five women employed in the lead pigments department submitted to surgical sterilization in order to retain their positions. A majority of the Commission held that "Congress did not intend the Act to apply to every conceivable aspect of employer-employee relations and that due to its unique characteristics this condition of employment is not a hazard within the meaning of the general duty clause." "Hazard" was defined to mean processes and materials that cause injury and disease by operating directly on employees as they engage in work or work-related activities. The Commission's decision was affirmed by the D.C. Circuit.[56]

In dissent, one Commissioner charged that the sterilizations resulted from a condition of employment imposed by the employer, and therefore should be considered a hazard subject to the general duty clause. Moreover, he cautioned that "[t]he exclusion of fertile women from certain employment invites employers to exclude other highly susceptible groups from employment when the effect varies among the exposed classes of individuals."

Even if an employer's reproductive health hazards policy were held to be within the purview of the general duty clause, it is not clear that a violation could be found. As discussed earlier, citation under § 5(a)(1) is inappropriate if a specific standard applies. An argument could be made that the "hazard" is not the employer's policy, but exposure to the hazard, specifically, lead. The employer's policy is simply the employer's attempt to deal with exposure to the hazard. Therefore, citation under the general duty clause is arguably precluded because of the existence of a standard dealing with lead that does not prohibit the employer's policy.

Another question is whether the Secretary would be able to prove all the necessary elements of a general duty clause violation. Specifically, the Secretary must specify the particular steps that the cited employer should have taken to avoid citation and to demonstrate the feasibility and likely utility of those measures. Simply ordering the return of the women to the toxic environment will not correct the problem of reproductive health hazards. Finally, an order directing the company to end its exclusionary policies would be prospective only and would not help the women already excluded or who had undergone sterilization.

Employee Duties.—Section 5(b) provides that "[e]ach employee shall comply with occupational safety and health standards and all rules, regulations and orders issued pursuant to this Act which are applicable to his own actions and conduct." Nevertheless, OSHA has no power to fine or otherwise sanction disobedient employees.[57] (A staff paper available from OTA discusses the leading case establishing this principle.)

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[53] Daniel International, Inc., 10 O.S.H. Cas. (BNA) 1557 (1982).

[54] OSHA Instruction CPL 2.50 (1982).

[55] American Cyanamid Co., 9 O.S.H. Cas. (BNA) 1596 (1981), aff'd, Oil, Chemical & Atomic Workers International Union v. American Cyanamid Co., 741 F.2d 444 (D.C. Cir. 1984).

[56] Id.

Final responsibility for employee compliance with OSHA's requirements rests with the employer. Therefore, employers must take every measure possible to ensure employee compliance, including the sanctioning of recalcitrant employees.

According to OSHA regulation, disciplinary measures taken by employers solely in response to employee refusals to comply with appropriate safety rules and regulations are not considered discrimination in violation of § 11(c) of the Act.[58] In fact, many collective bargaining agreements specifically require employee adherence to safety and health standards.

Decisions of the Commission have continued to hold that concerted employee refusal to comply is not a defense to a valid citation.[59] Employers have been found in violation even where a union contract prohibited employer discipline without going through the union foreman[60] and where prior attempts to enforce the standard had resulted in work stoppages up to 5 days long.[61] Since concerted employee refusal to comply with safety and health standards is not protected activity under the Act, employer disciplinary action is not prohibited.[62]

Procedures for Promulgation of Standards

Section 6(b) provides that any promulgation, modification, or revocation of OSHA standards must comply with specific rulemaking procedures.[63] Pursuant to § 6(b)(2), the Secretary is required to publish a notice of proposed rulemaking in the Federal Register and must allow 30 days after publication for interested parties to submit written data or comments. As a practical matter, OSHA usually allows at least 90 days for the submission of data or comments.[64]

OSHA usually schedules a public hearing when a proposal is issued, even though under § 6(b)(3) a hearing is not required unless requested. Most of the testimony time is used to question witnesses.[65] OSHA also produces its own witnesses and questions them.[66]

Hearings on proposed standards are of increasing importance, both in allowing interested persons an opportunity to present their views and in developing the record for subsequent judicial review. This may account for the great length of the hearings. For example, OSHA's first asbestos rulemaking hearing took 4 days and resulted in a record of 1,100 pages. The hearing on OSHA's carcinogens policy took 2 months and had a record of 250,000 pages.[67]

After the hearing is completed, the presiding administrative law judge usually gives the parties 30 days to submit additional data and 30 days after that to submit post-hearing briefs.[68] According to § 6(b)(4), the final standard (or a determination that no new standard is needed) must be issued within 60 days after the end of the comment period. For a variety of reasons, OSHA has rarely been able to meet this deadline.[69]

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[57] Atlantic & Gulf Stevedores, Inc. v. OSHRC, 534 F.2d 541, 553 (3d Cir. 1976).

[58] 29 CFR § 1977.22 (1984).

[59] Reinhardt's Plumbing & Heating, Inc., 5 O.S.H. Cas. (BNA) 1743 (1977); T. Clark & Son, 4 O.S.H. Cas. (BNA) 1913 (1976).

[60] Theodore D. Bross Line Construction Co., 3 O.S.H. Cas. (BNA) 1935 (1976).

[61] Weyerhaeuser Co., 3 O.S.H. Cas. (BNA) 1107 (1975).

[62] 29 CFR § 1977.22 (1984).

[63] OSHA rulemaking procedures appear at 29 CFR pt. 1911 (1984).

[64] B. Mintz, OSHA: History, Law and Policy 63 (1984).

[65] Id.

[66] Id. at 64.

[67] Id. at 62.

[68] Id. at 65.

[69] In National Congress of Hispanic American Citizens v. Usery, 554 F.2d 1196 (D.C. Cir. 1977), rev'g 425 F. Supp. 900 (D.D.C. 1975), the plaintiff sought an order requiring the Secretary to promulgate various agricultural standards. The district court granted summary judgment for the plaintiff and held that the timetable for promulgating standards in § 6(b) was mandatory. On appeal, the D.C. Circuit reversed, holding that the timetable was not mandatory because: 1) the Secretary was given discretion under §6(g) to "alter priorities and defer action due to legitimate statutory considerations"; and 2) inasmuch as the Secretary can decide not to issue a standard, "there is no sense in proceeding completely through the rulemaking process . . . only to end up with the Secretary issuing a notice that the standard is not adopted." On remand, the district court ordered the Secretary to complete development of a field sanitation standard as soon as possible and to submit a timetable for completion of the standard to the court within 30 days. National Congress of Hispanic American Citizens v. Marshall, No. 2142-73 (D.D.C. 1978). The D.C. Circuit again reversed. National Congress of Hispanic American Citizens v. Marshall, 626 F.2d 882 (D.C. Cir. 1979).

The final form of a standard may differ from the original proposal. Changes in a standard often reflect the comments and criticisms of interested parties as well as further agency deliberation and thus are to be encouraged. Nevertheless, the argument has been raised that where the final standard differs from the proposal, interested persons have been denied an opportunity to comment on the standard in its final form.[70]

Final OSHA standards typically contain detailed preambles, the standard itself, and any appendixes. A common format is as follows:[71]

1. an introductory discussion of the substance being regulated, its uses, and toxic properties;
2. a description of the background and history of the rulemaking proceeding;
3. a summary of the record and a discussion of the major issues raised by the proceeding—for health standards, this includes the extent of the risk from exposure to the substance, the PEL, and economic and technological feasibility;
4. a discussion of the specific provisions of the standard, section-by-section, including an explanation of why the particular provision was adopted and others were rejected;
5. a statement, as appropriate, on OSHA compliance with Executive orders on regulatory analysis,[72] the National Environmental Policy Act,[73] and the Regulatory Flexibility Act;[74] and
6. the text of the standard.

The validity of OSHA standards may be reviewed by a Federal appellate court if a petition is filed by an adversely affected party either before or after issuance of an OSHA citation. (Judicial review of OSHA standards is discussed in detail in a staff paper available from OTA.)

Possible Modification to Rulemaking Process

There is widespread agreement that the OSHA rulemaking process is slow, cumbersome, a drain on resources, and extremely adversarial.[75] In 1975, former Secretary of Labor John Dunlop attempted to expedite the process by using negotiations between the steel companies and unions to reach a consensus on a standard for coke oven emissions. "This effort failed, and Dunlop's approach was greeted with considerable hostility."[76]

In 1983, OSHA enlisted the services of neutral third-party mediators to facilitate a labor-industry agreement on revision of the existing benzene standard. Industry representatives from the Chemical Manufacturers Association, Rubber Manufacturers Association, American Iron and Steel Institute, and the American Petroleum Institute held a series of mediation sessions with union representatives from the AFL-CIO; United Steelworkers; Oil, Chemical, and Atomic Workers; and United Rubber Workers. Although it is not clear as yet whether mediation will be a success in the benzene standard, the use of mediation has prompted a discussion of the use of alternative dispute resolution techniques in OSHA rulemaking.[77]

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[70] Taylor Diving & Salvage Co. v. U.S. Department of Labor, 599 F.2d 622 (5th Cir. 1979); Daniel International Corp. v. OSHRC, 656 F.2d 925 (4th Cir. 1981).

[71] See B. Mintz, OSHA: History, Law and Policy 71 (1984).

[72] Exec. Order No. 12,291, 46 Fed. Reg. 13,193 (1981). See M. Rothstein, Occupational Safety and Health Law 71 (2d ed. 1983).

[73] 42 U.S.C. §§ 4321-4370 (1976 & Supp. V 1981); 29 CFR §§ 1999.1 to .8 (1984). See M. Rothstein, Occupational Safety and Health Law 70-71 (2d ed. 1983).

[74] 5 U.S.C. §§ 601-612 (1982).

[75] See, e.g., Comptroller General of the United States, Report to Congress, Delays in Setting Workplace Standards for Cancer Causing and Other Dangerous Substances (1977); General Accounting Office, Report to the Senate Committee on Labor and Public Welfare: Slow Progress Likely in Development of Standards for Toxic Substances and Harmful Physical Agents Found in Workplaces (1973).

[76] B. Mintz, OSHA: History, Law and Policy 88 (1984) (footnote omitted).

[77] Three senior OSHA officials were optimistic about mediation and thought that it could shorten the rulemaking process (both the hearing and comment period) and ease the resource drain of standards-setting. One thought that the best chance for success might be with chemicals that had not been the subject of prior regulation and where the positions of the parties had not hardened. He favored mediation to reach a draft standard and then allowing the public to comment.

Other former OSHA officials interviewed for this report were skeptical about mediation, perhaps as a result of OSHA's experience in 1975. One cautioned that it would be inappropriate to have the mediation take place too far along in the rulemaking process. Another former OSHA official, while agreeing that consensus is important, questioned whether OSHA can or should delegate its statutory responsibility to protect the public interest. Specifically, she questioned whether the unions can be expected to represent the views of all workers, including nonunion employees. A current OSHA official countered this argument by asserting that the regular comment period protects against this danger and permits comments by all concerned individuals.

Even those individuals who have doubts about mediation emphasize the need for labor-management cooperation. One former OSHA head recommends that labor and management attempt to reach agreement on key issues, while another former OSHA official notes that joint statements, stipulations of fact, and other agreements help the rulemaking process, but adds that such agreements are difficult to reach within the present rulemaking framework.

Emergency Temporary Standards

Section 6(c)(1) provides that if the Secretary determines that employees are "exposed to grave danger from exposure to substances or agents determined to be toxic or physically harmful or from new hazards," an emergency temporary standard (ETS) may be issued. These standards are effective immediately upon publication in the Federal Register without conforming to the detailed rulemaking requirements that apply to permanent standards. Under § 6(c)(3), an ETS may remain in effect for only 6 months; thereafter, the Secretary must promulgate a permanent standard under § 6(b). In this event the ETS serves as the proposed rule.[78]

An emergency temporary standard must be based on the existence of a grave danger[79] and the need for a standard to protect workers from the danger.[80]

Although emergency temporary standards need not be promulgated in accordance with the detailed procedures of § 6(b), certain procedural requirements must be complied with. One of these requirements is a statement of reasons, which must indicate:

1. the data in the record on which the ETS principally relies,
2. why those data suffice to show that the substances covered by the standard are harmful and pose a grave danger of exposure to employees, and
3. why the particular standard is necessary for the protection of employees.[81]

An ETS may be amended in the same manner as it was originally issued, according to the Fifth Circuit.[82]

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[78] See 29 CFR § 1911.12 (1984).

[79] According to the Third Circuit, the Act does not require an absolute certainty of the deleterious effect of a substance, but there must be evidence showing "more than some possibility" of a grave danger. Dry Color Manufacturers' Association v. U.S. Department of Labor, 486 F.2d 98, 104 (3d Cir. 1973). The dissent, however, contended that the purpose of the Act would be best effectuated by holding that even a scintilla of evidence can support an ETS. Id. at 110 (dissenting opinion).

The Fifth Circuit rejected the suggestion that deaths must occur before the issuance of an ETS. Nevertheless, the court held that there must be a danger of "incurable, permanent, or fatal consequences to workers, as opposed to easily curable and fleeting effects on their health. . . ." Florida Peach Growers Association v. U.S. Department of Labor, 489 F.2d 120, 132 (5th Cir. 1974).

In many instances, the only scientific research on a hazardous substance before promulgating an ETS will be animal studies. The application to humans of data extrapolated from animal studies of carcinogens was specifically accepted by the Third Circuit. Synthetic Organic Chemical Manufacturers' Association v. Brennan, 503 F.2d 1155 (3d Cir. 1974), cert. denied, 420 U.S. 973 (1975); Dry Color Manufacturers' Association v. U.S. Department of Labor, 486 F.2d 98 (3rd Cir. 1973). See generally McElveen and Eddy, Cancer and Toxic Substances: The Problem of Causation and the Use of Epidemiology, 33 Clev. St. L. Rev. 29 (1984); Comment, Judicial Attitudes Towards Legal and Scientific Proof of Cancer Causation, 3 Colum. J. Envtl. L. 344 (1977).

[80] The Third Circuit noted that the purpose of § 6(c)(1), to provide immediate protection, allows the Secretary to assume that employee exposure is occurring at any workplace containing the proscribed hazardous substance and where the corrective measures required by the ETS are not in effect. If the workplace is as safe and healthful without compliance with the letter of the ETS, the employer must resort to the variance procedures of § 6(d). Dry Color Manufacturers' Association v. U.S. Department of Labor, 486 F.2d at 102-03 n.3. Cf. Taylor Diving & Salvage Co. v. U.S. Department of Labor, 537 F.2d 819 (5th Cir. 1976) (stay of ETS granted where there was probability of success on merits of attack on standard and the likelihood of issuance of variance too uncertain to eliminate possibility of irreparable injury).

[81] In Dry Color Manufacturers' Association v. U.S. Department of Labor, 486 F.2d 98 (3d Cir. 1973), OSHA attempted to promulgate an ETS concerning exposure to 14 chemicals said to be carcinogens. The only statement of reasons was a conclusion, finding the chemicals to be carcinogens and reciting the need for a standard. The Third Circuit held that the statement of reasons was inadequate because it failed to meet the three-part test described in the text. The dissent in Dry Color, however, argued that preparing an exhaustive statement of reasons would be time-consuming and would render the ETS mechanism ineffective. Thus, it was suggested, all that should be required is notice of the Secretary's reason for issuing the ETS and access to the scientific data on which the Secretary relied. 486 F.2d at 110 (dissenting opinion). See also Synthetic Organic Chemical Manufacturers' Association v. Brennan, 506 F.2d 385 (3d Cir. 1974), cert. denied, 420 U.S. 973 (1975); Florida Peach Growers Association v. U.S. Department of Labor, 489 F.2d 120 (5th Cir. 1974); Associated Industries v. U.S. Department of Labor, 487 F.2d 342 (2d Cir. 1973).

[82] In Florida Peach Growers Association v. U.S. Department of Labor, organizations representing farmworkers contended that the Secretary exceeded his authority by summarily amending an ETS without using the modification procedures of § 6(b). The Fifth Circuit disagreed, observing that adherence to § 6(b) procedures could easily consume the entire 6-month life of the ETS. 489 F.2d 120 (5th Cir. 1974).

Box 7B.—The Problem of Emergency Temporary Standards

On November 4, 1983, OSHA promulgated an ETS for asbestos that lowered the permissible exposure limit (PEL). The ETS "emergency" was based on a new quantitative risk assessment showing that reducing the PEL for 6 months would save 40 to 80 lives. A group of asbestos products manufacturers sought judicial review of the ETS in the Fifth Circuit.

In *Asbestos Information Association* v. *OSHA*,[83] the Fifth Circuit held that the ETS was invalid and stayed its enforcement. The central theme of the court's analysis focused on whether OSHA had proven the need to adopt an ETS rather than modifying the existing standard after notice-and-comment rulemaking. The court pointed out that:

> . . . the plain wording of the statute limits us to assessing the harm likely to accrue, or the grave danger that the ETS may alleviate, during the 6-month period that is the life of the standard.[84]

One reason for publishing the ETS, according to OSHA, was to set in motion the process of promulgating a new permanent asbestos standard. The court was wary of permitting § 6(c) rulemaking to substitute for § 6(b) rulemaking.

The court rejected the asbestos manufacturers' argument that an ETS may not be issued unless it is based on new information. A "heightened awareness" based on new extrapolations certainly could justify the Secretary's action.[85] Nevertheless, the benefits of the ETS must outweigh its costs.[86] While it rejected the industry argument that the costs were excessive, the court was unconvinced of the accuracy of OSHA's estimate of the benefits.

Rather than rely on animal data, OSHA performed a detailed quantitative risk assessment and developed a dose-response curve from epidemiological studies of exposed workers. This assessment was made specifically to satisfy the "significant risk" requirement of the Supreme Court's *Benzene* decision[87] and the "grave danger" language of § 6(c). The Fifth Circuit was troubled by the possibility of inaccuracy in using risk assessment for a 6-month exposure period.

> [A]lthough risk assessment analysis is an extremely useful tool, especially when used to project lifetime consequences of exposure, the results of its application to a small slice of time are speculative because the underlying database projects only long term risks. . . . Applying the risk assessment process to a period of 6 months, one-ninetieth of OSHA's estimated working lifetime, only magnifies those inherent uncertainties.[88]

Moreover, as the court had previously noted, the mathematical extrapolations had not been the subject of "peer review."[89]

Finally, the court held that, even assuming OSHA's projected benefits would accrue from the ETS, OSHA failed to prove that an ETS—"the most dramatic weapon in its enforcement arsenal"—is necessary to achieve the projected benefits.[90] Specifically, OSHA had failed to enforce its current standard and could reduce exposures through enforcement and expeditious § 6(b) rulemaking.[91]

The court's opinion is subject to a variety of criticisms. Simply stated, the court is requiring OSHA to do the impossible. If the ETS were not accompanied by quantitative risk assessment of the expected benefits, undoubtedly the court would have held the ETS to be invalid. OSHA, however, performed a detailed risk assessment based on epidemiological evidence and calculated the number of lives expected to be saved. Differences of opinion over mathematical models should not obscure the fact that under any model a substantial number of lives would be saved by the ETS. It is never possible to predict precisely the effects of exposure on thousands of workers—nor is such evidence required. As the Supreme Court stated in the *Benzene* case:

> OSHA is not required to support its finding that a significant risk exists with anything approaching scientific certainty. Although the Agency's findings must be supported by substantial evidence, . . . a reviewing court [is required] to give OSHA some leeway where its findings must be made on the frontiers of scientific knowledge.[92]

Furthermore, the court's discounting of numerous reputable studies because of a lack of opportunity for public comment is antithetical to the express purpose of § 6(c).

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[83] 727 F.2d 415 (5th Cir. 1984).

[84] Id. at 422.

[85] Id. at 423.

[86] Id. at 423-24.

[87] Industrial Union Department v. American Petroleum Institute, 448 U.S. 607 (1980).

[88] 727 F.2d at 425-26.

[89] Id. at 421 n.15.

[90] Id. at 426.

[91] Id. at 427.

[92] Industrial Union Department v. American Petroleum Institute, 448 U.S. 607, 656 (1980).

Not surprisingly, both present and former OSHA officials interviewed for this report were dismayed by the court's decision and its implications. A former OSHA Chief stated: "You can kiss ETSs goodbye. They are not a viable option for the foreseeable future." Another former OSHA Chief did not agree that emergency standards are dead, citing DBCP, but cautioned that unless there were "hot new data" it would be best to use an ETS only for new hazards. Other former officials noted the problem of trying to persuade a reviewing court to uphold OSHA's use of an ETS to lower the PEL of a current standard, pointing out that even emergency standards for new hazards, such as hyperbaric diving, had been struck down.

Those interviewed stated that the record overwhelmingly supported issuance of the asbestos standard. According to an OSHA health standards official, "If there is no grave danger for asbestos, there is no grave danger for anything. The health effects of asbestos are 10 times worse than the rest of the substances combined." He added that, other than tobacco smoke, there were more epidemiological data on asbestos than any other substance of which he was aware. A former OSHA chief expressed a similar view. "The asbestos ETS was the best piece of work the agency had ever done—by far." A former DOL official reasoned that ETS challenges are difficult cases for the courts to decide on an emergency basis and that they are reluctant to order any capital expenditures when the life of the standard is only 6 months. In her view, Congress would need to amend § 6(c)'s "grave danger" language to make the ETS provision effective. In the meantime, two former OSHA heads agree that pursuing an ETS now would be a waste of the agency's limited resources in the sense of its very limited probability of being upheld.

Table 7-1.—Judicial Review of OSHA Emergency Temporary Standards

Standard	Date of enactment	Result	Reference
asbestos (I)	1971	Not challenged	
organophosphorous pesticides	1973	Vacated	Florida Peach Growers Association v. Department of Labor, 489 F.2d 120 (5th Cir. 1974)
14 carcinogens	1973	12 upheld	Dry Color Manufacturers Association v. Department of Labor, 486 F.2d 98 (3d Cir. 1973)
vinyl chloride	1974	Not challenged	
commercial diving	1976	Stayed	Taylor Diving & Salvage Co. v. Department of Labor, 537 F.2d 819 (5th Cir. 1976)
benzene	1977	Stayed	Industrial Union Department v. Bingham, 570 F.2d 965 (D.C. Cir. 1977)
DBCP	1977	Not challenged	
acrylonitrile	1978	Stay refused	Vistron v. OSHA, 6 O.S.H. Cas. (BNA) 1483 (6th Cir. 1978)
asbestos (II)	1983	Stayed	Asbestos Information Association v. OSHA, 727 F.2d 415 (5th Cir. 1984)

SOURCE: Office of Technology Assessment.

As box 7B demonstrates, OSHA has had a difficult time in the courts of appeals in challenges to its ETSs. This is particularly true in the Fifth Circuit, which has refused to uphold the ETS for pesticides, commercial diving, or asbestos.

Hazard Identification

The existence of health hazards is brought to OSHA's attention in three primary ways: 1) NIOSH brings its research to OSHA's attention, 2) advisory committees or consultants recommend health standards, and 3) citizens, labor unions, or companies petition OSHA or NIOSH for action. A discussion of NIOSH research appears in chapter 6. Advisory committees and citizen petitions are discussed below. (A detailed discussion of OSHA priorities in risk assessment and risk management appears in Appendix C.2.)

Standards Advisory Committees

Section 7(a) of the Act established a National Advisory Committee on Occupational Safety and Health (NACOSH) to advise the Departments of Labor and DHHS on matters related to the Act.[93] The Federal Advisory Council on Occupational Safety and Health (FACOSH) was established in 1974 to advise the Secretary of Labor on occupational safety and health matters relating to Federal Government employees.[94]

Between 1971 and 1976, most of the major health standards proposals were based on advisory committee recommendations. Since 1977, advisory committees have not been used to make recommendations. This change was based on detailed requirements for advisory committees mandated by OMB and the Carter Administration's effort to reduce the number of advisory committees. Instead, OSHA has used consultants to assist in the research and drafting of various parts of OSHA standards.[95]

Citizen Petitions

Section 6(b)(1) of the Act contemplates that information about the need for a new standard may be presented by "an interested person, a representative of any organization of employers or employees, a nationally recognized standards-producing organization, the Secretary of Health and Human Services, the National Institute for Occupational Safety and Health, or a State or political subdivision. . . ." The Secretary's regulations also provide that "any interested person may file . . . a written petition for the promulgation, modification, or revocation of a standard."[96]

Some citizen petitions have been granted by OSHA. For others, OSHA's refusal to issue an ETS or begin rulemaking on a permanent standard was sometimes followed by a court proceeding in which the petitioners sought to compel issuance of the standard. In some instances, such as pesticides, cotton dust, and labeling, the mere filing of the lawsuit may have been a substantial factor in issuing the standard more quickly.[97] In other instances, protracted litigation was necessary and had a mixed record of success for the petitioners.[98]

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[93] NACOSH is a permanent committee comprised of 12 members, 4 appointed by the Secretary of HHS and 8 appointed by the Secretary of Labor. The membership is comprised of representatives of management, labor, the public, and the occupational safety and health professions. NACOSH's basic purpose is to study all relevant material, consider possible alternatives, and weigh the feasibility of proposed standards.

[94] FACOSH is a permanent body, but is subject to renewal every 2 years. Exec. Order No. 12,196, 45 Fed. Reg. 12,769 (1980). The 16 members of FACOSH are appointed by the Secretary of Labor and serve staggered 3-year terms. Eight members are representatives of Federal agencies and eight members are representatives of Federal employee labor organizations.

[95] B. Mintz, OSHA: History, Law and Policy 65 (1984). Some present and former OSHA officials have differing views on the efficacy of advisory panels. An OSHA health standards official recommended amending the advisory panel language in the Act to eliminate the requirement of having representatives of various interest groups, and replace these members with independent and disinterested individuals. In his view, a panel of independent scientists could provide the peer review of technical documents needed by the agency. A former OSHA chief conceded that NACOSH has been "under-used and too political," but he still believes that it could perform the peer review function if it was seriously regarded by the Assistant Secretary of Labor for OSHA. Another former OSHA chief believes the committees are important, need not be nonpolitical, and benefit by having industry and employee representatives.

[96] 29 CFR § 1911.3 (1984).

[97] B. Mintz, OSHA: History, Law and Policy 197 (1984).

[98] Regardless of the merits of a citizen petition, the courts are extremely reluctant to order the issuance of a standard, particularly an ETS. The decision to issue a standard commits the agency to a substantial expenditure of resources and is often at the expense of other, arguably more important, rulemaking. Thus, in Public Citizen Health Research v. Auchter, the D.C. Circuit held that the district court erred in ordering OSHA to issue an ETS for ethylene oxide. 702 F.2d 1150 (D.C. Cir. 1983). While ruling that the district court "impermissibly substituted its evaluation for that of OSHA" in ordering the issuance of an ETS within 20 days, the court or-

Strategy for Hazard Exposure Control

Engineering Controls and Personal Protective Equipment

In its report, *Preventing Illness and Injury in the Workplace,* OTA examined the concept of "hierarchy of controls,"[99] in which the basic tenet is to control the hazard as close to the source as possible. In general, the order of controls is described as: engineering controls, work practice controls, and personal protective equipment. Sometimes administrative controls are included at the same order as either engineering controls or work practice controls. But in all cases, personal protective equipment is listed as the control of last resort. The problems of personal protective equipment arise out of: 1) limitations in performance; 2) difficulties in evaluating their performance; and 3) problems and burdens associated with their use, and the physical burdens they create.[100]

Engineering controls have the advantage of being easier to monitor to determine performance, are more reliable, enhance the development of new control and production technology, and do not create employee burdens. The main advantage of personal protective equipment is that it is usually significantly less expensive than engineering controls.

In February 1983, OSHA issued an advance notice of proposed rulemaking, stating its intention to reexamine its policy of giving priority to engineering controls.[101] In comments submitted to OSHA, employers and trade associations supported a change in OSHA policy to allow personal protective equipment to substitute for engineering controls. Comments from NIOSH, health and safety professionals working for universities and government agencies, and labor unions supported a continuation of OSHA's preference for engineering controls. In the preamble to the ethylene oxide standard, OSHA specifically restated the agency's policy of favoring the hierarchy of controls approach. (A discussion of the legal aspects of technological feasibility of OSHA health standards appears in Appendix C.3.)

Medical Removal Protection

OSHA's statutory authority to use medical removal protection (MRP) as a strategy for control was discussed earlier in this chapter. Assuming such authority exists, the next question is whether MRP is a viable strategy for control of reproductive health hazards.

The starting point for considering this issue is OSHA's lead standard. The standard set a PEL of 50 micrograms per cubic meter of air averaged over an 8-hour period and an action level of 30 micrograms. In addition, employees with blood-lead levels at or above 50 micrograms per 100 grams of whole blood (or who have symptoms of lead disease) are subject to medical removal.

In its preamble to the final lead standard, OSHA indicated that:

> To minimize the risk of genetic damage, menstrual disorders, interference with sexual function, lowered fertility, difficulties in conception, damage to the fetus during pregnancy, spontaneous miscarriage, stillbirth, toxic effects on the newborn, and problems with the development of the newborn or developing child, blood-lead levels should be kept below 30μg/100 g in both males and females exposed to lead who wish to plan pregnancies.[102]

Despite this language, the standard's PEL and MRP requirements contemplate that when full compliance is achieved the average blood-lead levels of workers will be 35 μg.[103] The OSH Act feasibility requirement, however, prevented OSHA from promulgating a stricter standard.[104] Reproductive effects were to be minimized, according to OSHA, by the action level, medical surveillance, and employee education.[105] Moreover, the stand-

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dered OSHA to expedite its rulemaking. Id. at 1153. In UAW v. Donovan, the district court, in refusing to order OSHA to issue an ETS on formaldehyde, stated: "Judicial review of an OSHA decision not to regulate is 'extremely narrow.' Reversal of OSHA's decision here thus requires the exceptional to exist from both 'substantive' and 'judicial review' perspectives." UAW v. Donovan, 45 O.S.H. Rep. (BNA) 2017 (D.D.C. July 12, 1984).

[99] U.S. Congress, Office of Technology Assessment, Preventing Illness and Injury in the Workplace (1985).

[100] Id.

[101] 48 Fed. Reg. 7474 (1983).

[102] 43 Fed. Reg. 52,960 (1978).

[103] Id. at 52,966.

[104] Id.

[105] Id.

ard's medical surveillance guidelines suggest that "the physician might recommend special protective measures or medical removal for an employee who is pregnant or who is planning to conceive a child. . . ."[106]

Can optional MRP under the lead standard prevent reproductive harms? Is optional or mandatory MRP for pregnant workers or for male and female workers attempting to parent children a feasible control strategy? The experts interviewed for this report were doubtful about MRP for a variety of reasons.

In many ways, lead is one of the best substances for medical removal because the effects of lead are largely reversible with discontinuation of exposure. But MRP as a reproductive health hazards control strategy, even for lead, is not entirely satisfactory. A NIOSH epidemiologist points out that there is a "rebound effect" of blood-lead levels after removal or chelation, where the levels will often go back up, without further exposure, after an initial drop. In addition, because of low calcium levels during pregnancy, lead stored in bones and other tissues may reenter the bloodstream. Finally, MRP would not prevent mutagenic effects that may have already occurred.

Although some individuals interviewed said that, in some situations, MRP could be a valuable strategy to use for substances other than lead, others expressed great reluctance to use MRP, mostly because of a lack of research on reproductive health hazards.

OSHA Reproductive Health Hazard Regulations

OSHA has only regulated three substances on the basis of their potential hazard to human reproductive health: DBCP, lead, and ethylene oxide, as discussed below.

DBCP

DBCP (1, 2-dibromo-3-chloropropane) is a liquid pesticide. In July 1977, workers at the Occidental Chemical Co. in Lathrop, California, noticed a pattern of infertility among DBCP workers. When tests were performed by Donald Whorton at the University of California, 14 of 38 workers tested had significantly reduced sperm counts.[107] No OSHA standard governing DBCP existed at that time.

In August 1977, the workers' union (OCAW) petitioned OSHA to issue an ETS for DBCP with a PEL of one part per billion (ppb). In September 1977, OSHA issued an ETS for DBCP, establishing an 8-hour TWA of 10 ppb and a 15-minute ceiling level of 50 ppb.[108] Based on evidence that DBCP was a carcinogen as well as a gametotoxin, in March 1978, OSHA issued a permanent standard lowering the 8-hour TWA to 1 ppb, with no ceiling limit.[109] Neither the ETS nor the permanent standard was challenged in court.

In addition to regulating the permissible airborne concentration of DBCP, the standard also prohibited dermal and eye contact, required exposure monitoring, established a respirator program, and provided for protective clothing, change rooms, and showers. The medical surveillance section of the standard provides for preplacement and annual examinations, which must include at least the following:

1. a medical and occupational history, including reproductive history;
2. a physical examination, including examination of the genito-urinary tract, testicle size, body habitus, and a determination of sperm count;
3. collection of a serum specimen, with the following determinations made by radioimmunoassay techniques utilizing National Institutes of Health specific antigen or one of equivalent sensitivity:
   a. serum follicle stimulating hormone,
   b. serum luteinizing hormone (LH), and
   c. serum total estrogen (females); and
4. any other tests deemed appropriate by the examining physician.[110]

The standard also provides for employee information and training as well as signs and labels.

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[106] 29 CFR § 1910.1025 app. C (1984).

[107] D. McCaffrey, OSHA and the Politics of Health Regulation 108 (1982).

[108] 42 Fed. Reg. 45,536 (1977).

[109] 43 Fed. Reg. 11,514 (1978) (codified at 29 CFR § 1910.1044 (1984)).

[110] 29 CFR § 1910.1044(m)(2) (1984).

In June 1979, the Environmental Protection Agency's Pesticide Advisory Committee recommended suspension of DBCP under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) because research by EPA and others demonstrated that DBCP caused cancer, harmful testicular effects, and genetic mutations in laboratory animals.[111]

EPA Administrator Doug Costle signed the notice of the order of emergency suspension for DBCP in July 1979, thereby beginning the 5-day period during which DBCP registrants could request administrative hearings on the order.[112] The hearings were held in October 1979. Extensive testimony was received, a significant portion of which supported EPA's original assessment of $42 million in production losses to growers. As a result of the cancellation hearings, EPA decided to suspend all uses of DBCP, with the exception of its use in Hawaiian pineapple fields, where residues were found not likely to occur given the method of DBCP application.[113] EPA's hearing concluded that "the immediate suspension of all uses of all registrations of pesticide products containing DBCP is necessary to prevent an imminent hazard."[114] In April 1981, EPA reached an agreement with the producers of the pesticide and canceled further administrative hearings. In that agreement, the Agency affirmed its 1979 decision banning DBCP for all uses except on Hawaiian pineapples.[115] OSHA's regulation of workplace exposure now has little relevance, except for those situations in which EPA granted exemptions[116] for the use of DBCP.[117]

In January 1985, EPA published a notice of its intent to cancel registration of DBCP used to fumigate Hawaiian pineapple fields, after finding DBCP contamination of groundwater.[118] The ban goes into effect in 1987.

Lead

Unlike the DBCP standard, which was promulgated largely because of the negative reproductive consequences of exposure, the lead standard was promulgated primarily to prevent other health problems (e.g., neurological disorders). Indeed, as discussed previously, the standard as promulgated is not sufficient to ensure that there will be no reproductive damage caused by exposure to lead, although it does attempt to minimize reproductive harms in several ways. These include medical removal provisions to protect workers wishing to have children.

The standard's medical surveillance section requires that a medical history be taken and must include a history of any reproductive problems. It provides that medical examinations, "if requested by an employee, shall include pregnancy testing or laboratory evaluation of male fertility."[119] The standard further provides that the employer must furnish a medical examination or consultation if the employee notifies the employer of a desire to obtain advice concerning the effects of current or past exposure on his or her ability

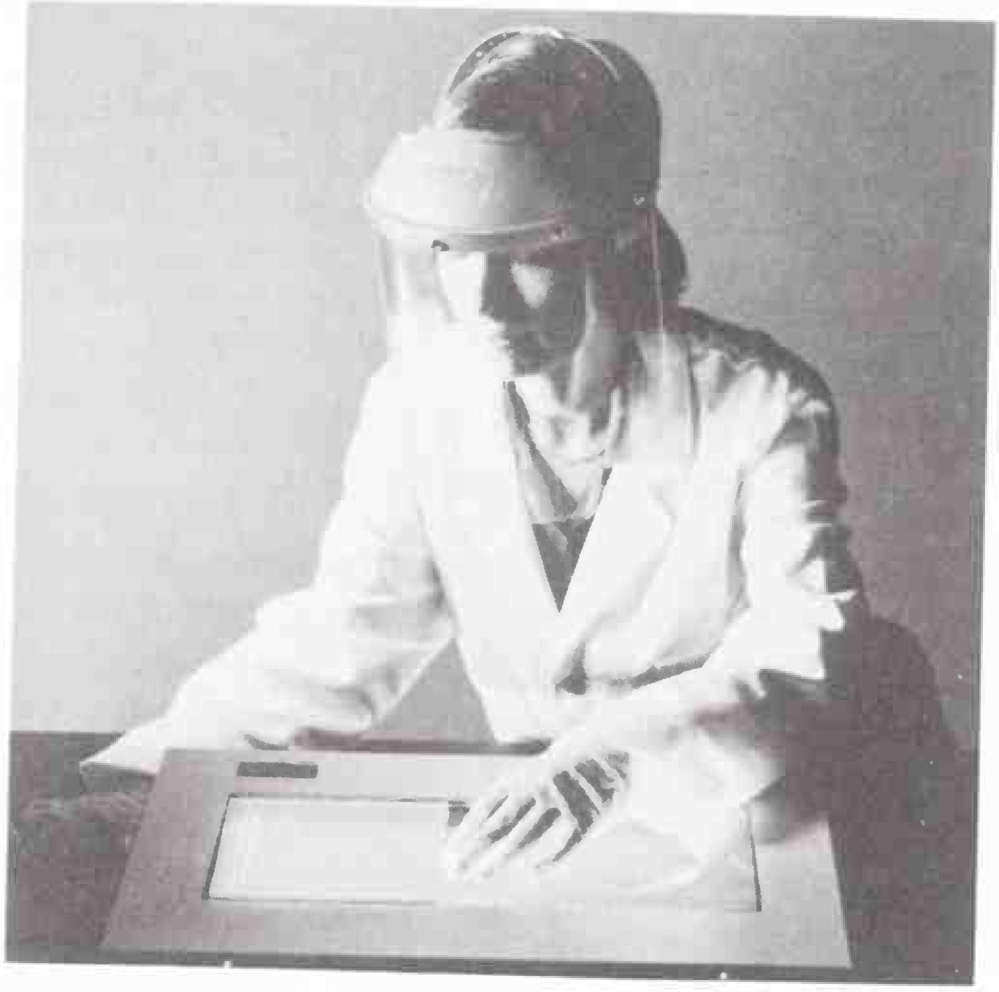

Photo credit: Pemina Meisels

Although the use of personal protective equipment is essential to many occupations, engineering, administrative, and work practice controls are given higher priority in efforts to limit hazard exposure.

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[111] For studies supporting suspension, see 44 Fed. Reg. 65,135 (1979).

[112] Chem. Reg. Rptr. (BNA) 577 (July 20, 1979).

[113] See 44 Fed. Reg. 65,135 (1979) (final suspension order). See also Chem. Reg. Rptr. (BNA) 1285 (Oct. 26, 1979) (cancellation hearings).

[114] Quoted in Chem. Reg. Rptr. (BNA) 1285 (Oct. 26, 1979).

[115] 46 Fed. Reg. 19,592, 19,596 (1981); Chem. Reg. Rptr. (BNA) 7 (Apr. 3, 1981).

[116] Exemptions can be granted under FIFRA Section 6d(C)(A), 7 U.S.C. § 136d(e)(A) (1982), if the Administrator determines that a use "will not have unreasonable adverse effects on the environment."

[117] Envtl. Health Newsletter, Oct. 1, 1982, at 3-4.

[118] 50 Fed. Reg. 112 (1985).

[119] 29 CFR § 1910.1025(j)(3)(ii) (1984).

to produce a healthy child.[120] A final relevant provision of the standard requires the employer to inform all exposed employees about the medical surveillance program, "including information concerning the adverse health effects associated with excessive exposure to lead (with particular attention to the adverse reproductive effects on both males and females)."[121]

Ethylene Oxide (EtO)

EtO is a clear, colorless gas that is used primarily as a chemical intermediate in the production of pesticides and as a sterilant and fumigant for hospital equipment. Because of EtO's use both as a pesticide as well as in nonfarm occupational settings, a controversy arose because the substance's use could be potentially regulated both by EPA under FIFRA and by OSHA under the OSH Act.

In 1978, citing multi-test studies demonstrating the mutagenic properties of EtO, EPA published a notice of Rebuttable Presumption Against Registration (RPAR) and placed EtO under special review.[122] The agency solicited comments on the action from registrants of the EtO pesticides and other interested parties. Pursuant to FIFRA, it requested that registrants submit data concerning the benefits of the compound that would justify its continued registration, as well as any further data on adverse health effects.

A number of studies released in 1981 and 1982, which showed additional evidence of the adverse effects of EtO, further fueled the controversy.[123] In January 1981, the Public Citizen Health Research Group and the American Federation of State, County, and Municipal Employees petitioned OSHA to force the agency to issue a new permissible exposure level for EtO.[124] They urged OSHA to establish an emergency temporary standard until a final regulation could be promulgated. The petition was denied and the group sued OSHA.[125] In January 1983, the U.S. District Court for the District of Columbia required OSHA to issue an emergency temporary standard by June 1983.[126] Additionally, it rejected OSHA's initial contention that EPA's actions precluded OSHA from taking regulatory action.[127]

A panel of the U.S. Court of Appeals for the District of Columbia overruled the lower court decision 2 months later.[128] The panel decided that the lower court had "impermissibly substituted its evaluation for OSHA's,"[129] and rejected the order requiring an emergency standard. Nevertheless, the D.C. Circuit directed OSHA to expedite completion of its ongoing rulemaking on EtO and within 30 days to promulgate a notice of proposed rulemaking.[130] However, the Court affirmed the lower court's decision on the question of jurisdiction over EtO. It stated:

> An easy question to resolve . . . is the Assistant Secretary's assertion that "there is a serious question as to OSHA's jurisdiction over hospital employees engaged in EtO sterilization activities," because of EPA's regulation of the chemical under the pesticide statute (the Federal Insecticide, Fungicide, and Rodenticide Act, 7 U.S.C. §§ 136-136y). OSHA, as the district court pointed out . . . has dealt with exposure to EtO for over a decade and has committed itself to eventual replacement of its dated standard. We agree entirely with the district court's conclusion that OSHA is not disabled from issuing an EtO standard in "areas—such as the health care industry—whereas EPA has apparently exercised minimal, if any,

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[120] Id.

[121] Id. § 1910.1025(j)(1)(v)(D).

[122] 43 Fed. Reg. 830 (1978).

[123] The "Bushy Run Study," released in February 1981, demonstrated that EtO caused cancer in laboratory animals at dosages as low as 10 ppm. Snellings, 1981, Final Report: Ethylene Oxide, Two Year Inhalation Study, Bushy Run Research Center Submission to EPA, Pittsburgh, PA.

In March 1982, a Johnson & Johnson study was released which showed chromosomal damage to hospital workers engaged in sterilization procedures using EtO. Preliminary report of Pilot Research Chromosome Study of Workers at Sites Where Ethylene Oxide Gas is Utilized as a Sterilant. Unpublished report available from Dr. J. Paul Jones, Director of Health Sciences, Johnson & Johnson, New Brunswick, NJ (1982).

In November 1982, the Hemminki study demonstrated a significantly higher rate of spontaneous abortion among hospital nurses using EtO in sterilizers. Hemminki, K., Mutinen, P., Saloniemi, I., Neimi, M.L., and Vainis, H., Spontaneous abortions in hospital staff engaged in sterilizing instruments with chemical agent. BRI. MED. I. 285: 1461-63 (1982).

[124] Public Citizen Health Research Group v. Auchter, 554 F. Supp. 242, 245 (D.D.C. 1983).

[125] Public Citizen Health Research Group v. Auchter, 554 F. Supp. 242 (D.D.C. 1983).

[126] Id. at 251.

[127] Id. at 250.

[128] Public Citizen Health Research Group v. Auchter, 702 F.2d 1150 (D.C. Cir. 1983).

[129] Id. at 1156-57.

[130] Id. at 1159.

> regulatory authority in an overlapping manner."[131]

In April 1983, OSHA published a Notice of Proposed Rulemaking for EtO that proposed to reduce the permissible 8-hour time-weighted average for EtO from 50 to 1 ppm.[132] A specific short-term exposure limit for EtO was not proposed, although comments on the issue were solicited.

In spite of OSHA's proposal, EPA published a Notice of Revised Labeling for Certain Pesticides containing EtO in April 1984.[133] EPA stated in that notice that:

> . . . the evidence of the mutagenicity of EtO has continued to accumulate and the Agency believes that EtO poses a mutagenic risk to exposed humans. . . . New evidence also augments the concern that EtO may produce adverse reproductive effects.[134]

The notice makes clear that the agency considers the use of EtO in hospitals to be a pesticidal use, and states:

> (T)he changes contained in this notice are limited to hospital and health care facility use. . . . (T)he Agency decided to focus on this use first because hospital and health care facility workers are the single largest group of workers exposed to EtO and are believed to be occupationally exposed to the highest levels of EtO.[135]

EPA proposed product label changes requiring modifications in workplace design and practice in hospitals and health care facility to control exposure to EtO.

The 1984 Federal Register notice also addresses the progress of the special review on ethylene oxide that EPA initiated in 1978.[136] It states that EPA "intends to pursue the comprehensive evaluation of all EtO data and, upon completion of this evaluation, to issue a Preliminary Notice of Determination Concluding the RPAR [Special Review] Process."[137] The rationale supplied by EPA for the interim label changes was "because it will take additional time to develop the final position on all EtO uses and it is evident" from available information that exposure limitations should be implemented as soon as practical.[138]

EPA also completed a review draft health assessment for ethylene oxide under the Clean Air Act in April 1984.[139] Although the assessment was initially developed for evaluating EtO as a hazardous air pollutant under § 112 of the Clean Air Act, the scope of the assessment was expanded to address multimedia impacts. EPA concluded on the basis of its draft review that ethylene oxide produces developmental toxicity in laboratory animals when conducted at or near maternal toxic doses (maximum tolerated dosages), and produces adverse reproductive effects and testicular atrophy at levels lower than those which produce general toxicity. It also concluded that EtO may cause spontaneous abortions to hospital personnel in occupational settings and is capable of causing gene mutations.

Still under the D.C. Circuit court order to produce a permanent standard, OSHA published a final standard consisting of an 8-hour TWA occupational exposure level for EtO of 1.0 ppm and an action level of 0.5 ppm in June 1984.[140] Reportedly under pressure from the Office of Management and Budget, OSHA sidestepped the issue of a short-term exposure level by saying it would consider the issue at a later time.[141] The Public Citizen Health Research Group immediately filed suit, claiming that OSHA had violated the court settlement by failing to propose a short-term exposure level.

On the same day that OSHA published its standard, EPA withdrew the labeling standard it had proposed in April 1984.[142] The rationale for the action was that EPA did not want to preempt OSHA's ability to set a comprehensive (long- and short-term) standard for exposure to EtO in hospitals and health care facilities. The agency stated:

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[131] Id. at 1156 n.23. In another late unrelated action concerning EPA's consideration of EtO, the Natural Resources Defense Council (NRDC) and the AFL-CIO filed suit against EPA in May 1983 charging that the Agency had conducted ex-parte meetings with industry to terminate RPARs for certain pesticides, including EtO. The groups charged that the public had been illegally excluded from the decisionmaking process. This contention is presently being reviewed in settlement negotiations.

[132] 48 Fed. Reg. 17,284 (1983).

[133] 49 Fed. Reg. 15,628 (1984).

[134] Id.

[135] Id.

[136] Id.

[137] It is unclear how this process may affect future EPA regulatory action on EtO.

[138] 49 Fed. Reg. 15,628 (1984).

[139] EPA-600/8-84-009A (April 1984).

[140] 49 Fed. Reg. 25,734 (1984).

[141] Occupational Health and Safety Letter, at 5 (June 22, 1984).

[142] 49 Fed. Reg. 25,675 (1984).

> Since the issuance of the notice, substantial concern has been raised over the possibility that adoption of the requested labeling changes, which are intended to affect workplace design and practice in hospitals and health care facilities, might have a preemptive effect on OSHA's ability to set comprehensive EtO standards. EPA has determined that it would be prudent to withdraw its April 18, 1984 notice and the associated requests that registrants submit revised labeling for pesticide products containing EtO. [143]

According to one union lobbyist, union pressure was responsible for persuading EPA to withdraw the labeling standard because the union believed that the proposal would interfere with OSHA's issuance of a short-term exposure limit for EtO and with implementation of the standard.[144] An EPA staffer with responsibility for the special review of EtO said that the agency was unsure of whether or not it would take further action on EtO. [145]

In response to an order from the U.S. Court of Appeals for the District of Columbia, in August 1984, OSHA presented a sworn affidavit in Federal district court stating that it would complete a rulemaking on a short-term exposure limit for EtO by December 1984.[146] In December of that year, OSHA informed the district court that adoption of a short-term exposure limit for EtO was not warranted by the available evidence and was therefore not appropriate for inclusion in the final standard.[147] The statement of reasons was published in January 1985.[148]

The current OSHA standard not only lowered the PEL, but included other measures designed to protect the reproductive health of workers. Some of these measures are identical to the lead standard's requirements, and some are slightly different. As in the lead standard, employers must provide a medical examination or medical consultation for employees desiring information about the effects of current or past exposures on the ability to produce a healthy child.[149] As with lead, the medical history also includes a reproductive history.[150] The physical examination must also give particular attention to the reproductive system.[151] Pregnancy and fertility testing must be provided if the employee so requests, but only if the physician concurs in the need for testing.[152] The preamble to the standard explains that the purpose of requiring the physician's concurrence for pregnancy or fertility testing is to avoid "abusive or frivolous" requests, although OSHA cited no evidence of such abuses under the lead standard.

The ethylene oxide standard requires the use of warning signs and labels, which must clearly note that ethylene oxide is a cancer hazard and a reproductive hazard. Employees also must be given training and information concerning ethylene oxide use, including the substance's potential for reproductive harm.

Other Reproductive Health Hazards

OSHA standards have set PELs for a number of other known or suspected reproductive health hazards, including benzene, cadmium, mercury, and ionizing radiation. The scientific evidence relating to these agents is discussed in chapter 4. No efforts have been specifically addressed to preventing reproductive harms from exposure to these hazards. (Most of these standards are those adopted under section 6(a) of the OSH Act when OSHA was first created.)

An OSHA official has observed that regulation of reproductive hazards is constrained by the paucity of studies on reproductive health effects of substances found in the occupational setting:

> We're no better off today in terms of studying reproductive health hazards than we were

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[143] Id. at 25,675.

[144] Personal communication with Jordan Barab, AFCSME (Sept. 10, 1984).

[145] Personal communication with Ann Barton, Deputy Division Director, Hazard Evaluation Division, EPA (Sept. 11, 1984).

[146] Chem. Reg. Rptr. (BNA) 608 (Sept. 14, 1984).

[147] 14 O.S.H. Rep. (BNA) 563 (Jan. 3, 1985).

[148] 50 Fed. Reg. 64 (1985). The Public Citizen Health Research Group has filed suit challenging OSHA's decision to exclude a short-term exposure limit for EtO from OSHA's final EtO standard. Public Citizen Health Research Group v. Rowland, Nos. 84-1252, 84-1392 (D.C. Cir. filed January 1985).

[149] 29 CFR § 1910.1047(j)(2)(i)(E) (1984).

[150] Id. at § 1910.1047(j)(2)(ii)(A)(1).

[151] Id. at § 1910.1047(j)(2)(ii)(A)(2).

[152] Id. at § 1910.1047(j)(2)(ii)(B).

> in the 1950s. However, in terms of regulating hazards, we're worse off because we've done little or nothing to contain substances shown to be teratogenic to humans exposed in the occupational setting.

A former NIOSH director, agreeing that the toxicology has not been well developed, added that "traditional teratological studies strengthened the stereotype of the exclusively maternal role in the transmission of reproductive health harms."

Several of the NIOSH criteria documents submitted to OSHA have identified reproductive health hazards appropriate for regulatory action. These hazards include antimony,[153] carbon disulfide,[154] ethylene thiourea,[155] polychlorinated biphenols (PCBs),[156] and nitrous oxide.[157] Formaldehyde[158] and ethylene dibromide (EDB),[159] the subjects of recent citizen petitions, have also been linked with reproductive health harms.

Generic Standards

As discussed in this report, the **promulgation of new OSHA standards is a long, costly, and difficult process. In reviewing OSHA standards, the courts insist on procedural regularity, a showing of significant risk, the use of the "best available evidence," proof of material impairment, demonstration of technological and economic feasibility, and substantial evidence of other crucial elements. These requirements, along with budget and personnel problems, legal challenges, policy shifts at OSHA, and other factors have resulted in very few new standards being promulgated.**

There have been only 10 successful permanent rulemaking actions since 1971, resulting in 22 health standards. The bulk of OSHA health standards remain the outdated (1968) American Conference of Government Industrial Hygienists' threshold limit values adopted by OSHA in 1971. The standards contain mostly PELs, with no requirements for environmental monitoring, biological monitoring, or medical surveillance. Hundreds of new chemicals are being introduced into industry each year, but few new standards are being promulgated. The agency is always "playing catch-up." For example, in 1977 OSHA lowered the PEL for the pesticide DBCP when it was shown that DBCP was a gametotoxin and carcinogen. The pesticide often used as a substitute for DBCP is EDB, a potent carcinogen that also has been linked to a variety of reproductive health harms. OSHA is now examining restrictions on exposure to EDB.

During the Ford and Carter Administrations, OSHA attempted to promulgate health standards on a "generic" basis. That is, OSHA sought to establish a regulatory framework for rulemaking on an entire class of substances or hazards on a single occasion.[160] It was hoped that such an approach would result in more efficient and expeditious promulgation of standards. The "standards completion project," begun in 1974, was a generic rulemaking project that attempted to update the original health standards package. The generic carcinogen policy developed criteria and procedures for regulating carcinogenic substances. Both efforts failed: the standards completion project was abandoned and the generic carcinogen policy, still pending in the courts, is still in effect but has not been relied on by the current Administration. Although generic-type rulemaking has produced the access to employee exposure and medical records standard and the hazard communication standard, there have been no further efforts to promulgate generic standards.

The broad array of reproductive health hazards to be regulated raises the question of whether it is possible or desirable to promulgate a generic reproductive health hazards standard. A former OSHA Director, who considers it possible, recommends coordinating various regulatory agencies (e.g., OSHA, EPA, Mine Safety and Health Administration, Consumer Product Safety Commission, Food and Drug Administration) and starting with a less controversial generic standard before moving to reproductive health hazards. A former NIOSH chief agrees with the idea of beginning with a simpler generic standard, such as skin irritants, but points out the difficulties of proposing a generic standard for reproductive health hazards given the paucity of information. Three other high-ranking officials also support the idea of a generic approach to reproductive health hazards.

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[153] NIOSH No. 78-216.

[154] NIOSH No. 78-166.

[155] NIOSH No. 77-140.

[156] NIOSH No. 77-156.

[157] NIOSH No. 78-144.

[158] NIOSH No. 77-225.

[159] NIOSH No. 76-149.

[160] For a further discussion, see B. Mintz, OSHA: History, Law, and Policy 82-86 (1984).

A key issue in using such an approach is deciding on the quantity and quality of data needed before specific standards can be issued. One NIOSH official stated that "we need to protect workers on the basis of toxicological studies, rather than waiting for epidemiological data," while another questioned whether we know enough about the physiological processes of reproductive health harms to use a generic approach.

EEOC and OFCCP Proposed Interpretive Guidelines on Employment Discrimination and Reproductive Health Hazards

OSHA's attempts to regulate reproductive health hazards have invariably raised employment discrimination issues. For example, in the *American Cyanamid* case,[161] discussed earlier, OSHA unsuccessfully attempted to use § 5(a)(1) to prohibit an employer's policy of excluding all fertile women from working where there was exposure to lead.

In January 1980, the Equal Employment Opportunity Commission (EEOC) and the Department of Labor's Office of Federal Contract Compliance Programs (OFCCP) issued joint Proposed Interpretive Guidelines on Employment Discrimination and Reproductive Health Hazards.[162] The Guidelines, issued pursuant to Title VII of the Civil Rights Act of 1964 and Executive Order 11,246, were proposed to address the fact that:

> . . . an increasing number of employers and contractors . . . are initiating policies excluding all women of childbearing capacity from certain jobs because of exposure to hazardous substances or conditions.[163]

The Proposed Guidelines would have permitted the "temporary emergency exclusion" of only male, female, or pregnant employees under limited circumstances where there is proof of a hazard to one sex or to the future offspring of one sex, but not to the other sex and where no other alternatives were available. The Guidelines did not, however, address the issue of how the emergency exclusion would be triggered. For example, there was no discussion of whether an employer could have required women employees to take periodic pregnancy tests.

The Guidelines would have prohibited altogether any reproductive health hazard policies applicable to only one sex. Facially neutral policies that have an adverse impact on one sex were to be justified "in accordance with relevant legal principles." (Presumably, this meant establishing a business necessity or job-relatedness defense, as discussed in chapter 8.)

The proposal evoked widespread controversy. In January 1981, the Proposed Guidelines were withdrawn,[164] largely, according to a former chairperson of the EEOC, as a result of a lack of consensus on the scientific evidence received in response to the proposal, without which it was considered virtually impossible to issue a final regulation dealing with this complex and controversial subject.

The Proposed Guidelines contemplated active "consultation and coordination" between EEOC, OFCCP, NIOSH, and OSHA. Several present and former OSHA officials interviewed for this report had reservations about such OSHA involvement, asserting that OSHA lacked the statutory authority, resources, or expertise to become involved in discrimination claims. A former OSHA chief, who was instrumental in getting the proposed guidelines issued, disagreed. In her view, OSHA has "inherent responsibility" in this area; and should lend technical support and assistance to EEOC and NIOSH. Neither OSHA, EEOC, nor OFCCP currently plan to reconsider rulemaking in this area. However, EEOC and OSHA will continue to handle allegedly discriminatory employment policies relating to reproductive health on a case-by-case basis.

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[161] American Cyanamid Co., 9 O.S.H. Cas. (BNA) 1596 (1981), aff'd, Oil, Chemical & Atomic Workers International Union v. American Cyanamid Co., 741 F.2d 444 (D.C. Cir. 1984).

[162] 45 Fed. Reg. 7514 (1980).

[163] Id.

[164] 46 Fed. Reg. 3916 (1981). The EEOC's statement accompanying the withdrawal indicated that cases of discriminatory FPPs would continue to be monitored and evaluated under existing Title VII principles.

Hazard Communication Standard

In November 1983, OSHA issued its final hazard communication standard, after nearly a decade of study and proposed rulemaking.[165] A former OSHA chief called the regulation "the single most significant and far-reaching standard ever written by this agency."[166] The regulation covers approximately 15 million workers[167] and is expected to cost $600 million.[168] It requires chemical manufacturers and importers to assess the hazards of the chemicals they produce or import, and communicate that information to workers. Furthermore, distributors of hazardous chemicals must label chemical containers, and provide a material safety data sheet to customers in the manufacturing sector (SIC codes 20-39).

In January 1977, OSHA issued an advance notice of proposed rulemaking on chemical labeling.[169] After receiving comments from State and local government agencies, businesses, and labor organizations in favor of apprising workers of health hazards caused by exposure to chemicals, OSHA published a notice of proposed rulemaking in January 1981.[170] The 1981 proposal was in most respects more comprehensive and costly than the regulation that was eventually enacted. The proposal would have required chemical hazard labeling on all containers (and pipes) used in the workplace, in addition to labeling by distributors who ship chemical containers to manufacturers,[171] and would have covered approximately 20 million workers,[172] whereas the present regulation covers roughly 15 million workers.[173]

Less than a month after the rule was proposed, it was withdrawn by the Reagan Administration. Due to a growing awareness of the importance of the issue, and perhaps as a result of Federal inaction in this area, several States enacted labeling and disclosure laws.[174]

OSHA then revised its proposal and issued another notice of proposed rulemaking. The 1983 Hazard Communication Standard is the culmination of OSHA's activities in this area. (The coverage of employees, employers, and chemicals in the standard is described in Appendix C.4.) OSHA's regulation notwithstanding, numerous State legislatures seeking more stringent regulation of chemical health hazards have continued to enact "right-to-know" laws. For example, New Jersey, which produces approximately 25 percent of all chemicals manufactured in the United States,[175] passed a law in 1983 that is considerably broader than the OSHA regulation.[176] As of April 1985, 20 States had passed such statutes (see table 7-2), and the District of Columbia, Georgia, Louisiana, Missouri, North Carolina, and Texas are considering passage of right-to-know laws.[177] Whether these laws are preempted by the OSHA standard is under judicial review.

Table 7-2.—States With Right-to-Know Laws

State	Effective date	State	Effective date
Alaska	1983	Michigan	1980
California	1983	Minnesota	1983
Connecticut	1983	New Hampshire	1983
Delaware	1985	New Jersey	1983
Florida	1985	New York	1980
Illinois	1984	Oregon	1984
Iowa	1984	Pennsylvania	1985
Maine	1980	Rhode Island	1983
Maryland	1984	West Virginia	1981
Massachusetts	1984	Wisconsin	1982

SOURCE: Himmelstein and Frumkin, "The Right-to-Know About Toxic Exposures," *New Eng. J. Med.* 312(11):688, Mar. 14, 1985.

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[165] 29 CFR §1910.1200 (1984).

[166] Chemical Right-to-Know Requirements: Federal and State Laws and Regulations on Disclosure, Special Report (BNA) 3 (1984) [hereinafter cited as BNA Special Report.]

[167] Workers' "Right-to-Know": OSHA's Hazard Communication Rule, Issue Brief IB84103, The Library of Congress Congressional Research Service, 2 (1984) [hereinafter cited as CRS.] Another authority has estimated that approximately 14 million workers will be protected by the OSHA Regulation. BNA Special Report, supra note 166, at 1.

[168] Preamble to final OSHA Standard on Workplace Hazard Communication, O.S.H. Rep. (BNA) 700, 748 (Dec. 1, 1983) [hereinafter cited as BNA Preamble.]

[169] 42 Fed. Reg. 5372 (1977).

[170] 46 Fed. Reg. 4412 (1981).

[171] BNA Preamble, supra note 168, at 701. The 1981 regulation contained no employee training requirement, whereas the present OSHA regulation does.

[172] BNA Special Report, supra note 166, at 1.

[173] CRS, supra note 167 and accompanying text.

[174] By 1981, Maine, Michigan, New York, and West Virginia had enacted right-to-know laws.

[175] CRS, supra note 167, at 6.

[176] BNA Special Report, supra note 166, at 18. New Jersey's law requires that employees in nearly all workplaces be informed of the health hazards of approximately 2,000 chemicals.

[177] Status of Right-to-Know Legislation, Women's Occupational Health Resource Center News, Columbia University 1, 5 (Aug. 1984). See also BNA Special Report, supra note 166, at 1; personal communication, Peg Seminario, Assistant Director, Department of Occupational Safety, Health, and Social Security, AFL-CIO (Apr. 12, 1985).

Reproductive Health Hazards.—Chemicals posing potential reproductive health hazards are not expressly addressed by OSHA's regulation, though they are implicitly covered. However, many State right-to-know laws explicitly discuss reproductive health hazards,[178] and some States' statutes that do not have taken the position that such hazards are implicitly covered within the State statute's definition of toxic and hazardous substances.[179] In addition to specifically listing teratogens as a class of hazardous chemicals regulated by their laws, two States have special trade secret provisions for teratogens. Massachusetts requires that containers of chemical teratogens, the compositions of which are trade secrets, be labeled with a large "T" at the worksite. And New Jersey denies all trade secret protection to teratogens.

Similar concern about protecting workers from reproductive health hazards was expressed by Connecticut's right-to-know law, which contains a nondiscrimination provision. Connecticut's law prohibits the sterilization of employees as a condition of employment, transfer, or promotion.[180] The law also protects female employees by requiring an employer to attempt to offer to transfer pregnant employees when the employer or employee reasonably believes that continued exposure will threaten her reproductive health, or the health of her offspring.[181]

Disclosure: Written Hazard Communication Program.—If a chemical manufacturer, importer, or distributor determines that a substance poses a hazard to workers, a written hazard communication program must be developed.[182] Three methods of communicating information are required by the Act: 1) labeling, 2) supplying material safety data sheets (MSDSs), and 3) employee information and training programs.

Trade Secrets.—One of the most controversial provisions of OSHA's regulation is the section dealing with trade secrets. The standard permits chemical manufacturers or importers to withhold the chemical name and other information about the chemical from the MSDS if the manufacturer or importer believes the information is a trade secret.[183] While the chemical name and other data may be withheld, information concerning the hazards of the chemical must be disclosed. In medical emergency situations, the employer must disclose the chemical name; in nonemergency situations, however, an employer claiming a trade secret need only disclose the identity of a substance to medical personnel if several conditions are first met.[184]

The trade secret provision of OSHA's regulation has been subject to strong criticism. Critics maintain that too much discretion is conferred on employers in determining what constitutes a trade secret and that challenging an employer's decision to withhold information is costly, cumbersome, and time-consuming.[185] Critics also contend that OSHA's review of an employer's claim of a trade secret is too limited.[186]

State hazard communication laws regulating trade secrets vary in at least one significant way from OSHA's regulation. Most States automatically review the determination of a trade secret made by an employer.[187]

Preemption.—The preemption doctrine—based on the supremacy clause of the U.S. Constitution[188]—holds that State laws which conflict with Federal laws that constitutionally regulate the same subject matter are invalid. OSHA maintains that all aspects of State right-to-know laws that have not received prior approval by OSHA are preempted by OSHA's Hazard Communication rule, except those aspects pertaining to community information requirements.[189] Yet constitutional concerns, State interests that the OSHA standard cannot regulate (e.g., protection of State public employees), and the language of the OSH Act itself require that a more detailed analysis of the preemption issue be undertaken by the courts.

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[178] E.g., Alaska, Connecticut, Illinois, Minnesota, Maine, Massachusetts, and New Jersey expressly mention reproductive hazards in their right-to-know laws.

[179] Personal communication, Ivan Russell, Minnesota OSHA (January 1984); Richard Stone, New York Bureau of Toxic Substances (January 1984).

[180] Conn. Gen. Stat. Ann. § 31-40h (Supp. 1984).

[181] Conn. Gen. Stat. Ann. § 46a-60(a)(7)(E) (Supp. 1984).

[182] 29 CFR § 1910.1200(e) (1984).

[183] 29 CFR § 1910.1200(i) (1984).

[184] 29 CFR § 1910.1200(i)(3) (1984).

[185] CRS, supra note 167, at 5.

[186] Personal communication with Peg Seminario, Assistant Director, Department of Occupational Safety, Health, and Social Security, AFL-CIO (Feb. 21, 1984).

[187] California, Connecticut, Illinois, Massachusetts, Minnesota, New Hampshire, New Jersey, New York, and Wisconsin have automatic review provisions.

[188] U.S. Const. art. VI, § 2.

Conclusions

The Occupational Safety and Health Administration has authority to regulate occupational reproductive health hazards in various ways. The agency can promulgate permanent health standards concerning a single hazardous substance, a group of specific substances, or even reproductive health hazards as a class, after extensive and cumbersome rulemaking proceedings that may take several years to complete. OSHA has promulgated permanent standards for only three substances—DBCP (1,2-dibromo-3-chloropropane), lead, and ethylene oxide—that include specific guidelines for the protection of reproductive health.

As detailed in the text of this report, promulgating any new OSHA health standard is extremely difficult. It depends on a good working relationship between NIOSH and OSHA, adequate budgets and personnel for each agency, and insulation of the decisionmakers from the political pressures that invariably arise when new regulations are proposed. The rulemaking process is protracted, detailed, cumbersome, resource-draining, and adversarial. The reviewing courts have required detailed analyses of significant risk, technological feasibility and economic feasibility. The courts also have shown a reluctance to uphold the validity of emergency temporary standards, and have required, at times, precise and almost cataclysmic evidence of "grave danger."

The prospects are unclear for new standards or more stringent modifications of existing standards to protect reproductive health. A number of problems exist. Scientific evidence concerning reproductive health hazards in the workplace is lacking, in part, because of a historical lack of interest in this field at OSHA, NIOSH, CDC, and PHS. There are also problems with methodologies for new studies, such as the need to develop better models for extrapolating animal data to humans, the ongoing problem of selection of proper controls, and the lack of large enough study populations for epidemiological studies.

The prospect of new substances being introduced at a faster rate than regulations are currently being issued has raised the question of whether a generic reproductive hazard standard is possible or feasible. Such a policy would establish the framework for regulating a variety of substances and would, presumably, allow for more efficient and expeditious standards promulgation. Although many individuals interviewed supported the idea in principle, there are potential scientific, legal, and political stumbling blocks.

OSHA may issue an emergency temporary standard (ETS), effective immediately, if it determines that employees are exposed to a "grave danger" from exposure to health hazards. No court has decided whether reproductive health problems are "grave dangers," though a recent Federal court of appeals decision suggests that only "incurable, permanent, or fatal" health consequences could support the issuance of an ETS. Since OSHA has lost several challenges in the courts of appeals to its ETSs, OSHA is unlikely to issue ETSs for known or suspected reproductive health hazards, especially in situations where the reproductive damage is temporary.

Even where no temporary or permanent health standards apply, OSHA is empowered to ensure that employers are fulfilling their general duty under the OSH Act to furnish working conditions free from "recognized hazards" that are likely to cause death or serious physical harm. Since a hazard is considered recognized only if it is common knowledge in the employer's industry or if the employer had actual or constructive knowledge of the hazard, it may be difficult for OSHA to prove that newly documented or suspected reproductive health hazards are recognized. The general duty clause is therefore unlikely to be a substitute for an emergency temporary standard or to serve as an interim measure until a permanent standard is enacted.

It is unclear whether OSHA has authority to address the problem of reproductive health hazards by regulating the employer's options relating to employee exposure, such as employment policies that exclude women from jobs involving potentially hazardous exposures. The Occupational Safety and Health Review Commission has ruled that Congress did not intend OSHA to have authority to issue a citation to an employer whose fetal protection policy excluding fertile women from certain jobs resulted in several women submitting to surgical sterilization to keep their jobs. The Commission's decision has been affirmed by the D.C. Circuit.

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[189] Personal communication with Jennifer Silk, Health Scientist, OSHA (Mar. 29, 1985). See also CRS, supra note 167, at 6.

Even if OSHA had the authority to expedite the permanent health standard procedure or to enact ETSs without fear of being reversed in court, it is not clear that health standards for reproductive health hazards would result. This is attributable both to the difficulty of identifying these substances and to less-than-ideal working relations between OSHA and NIOSH resulting from the personal relations, policies, and perceptions of their leaders. OTA conducted interviews with many present and former OSHA and NIOSH officials to explore the agencies' relations and coordination with respect to occupational health issues in general and reproductive health hazards in particular. The institutional concerns, priorities, and policies of OSHA and NIOSH often vary considerably, with officials of each agency indicating disapproval of the priorities and policies of the other. Interagency cooperation also varies with the political philosophy of the Administration in power. Under the Carter Administration, OSHA and NIOSH developed a close working relationship, including personnel exchanges and various joint programs, though this resulted in criticism of NIOSH for allegedly abandoning its neutrality. The Reagan Administration, which believes in the clear separation of research from regulation, has discontinued some cooperative programs. Interviews revealed that a 1979 interagency agreement concerning cooperative programs between NIOSH and OSHA was unknown to many current, high-ranking OSHA officials.

In addition, OSHA has a shortage of professional technical staff to develop health standards, and this staff shortage may result in insufficient technical expertise for evaluating NIOSH's work and taking appropriate regulatory actions. Adding technical staff would likely require additional legal and administrative staff to direct and implement a regulatory strategy for reproductive and other health hazards.

EPA AUTHORITY TO ADDRESS REPRODUCTIVE HEALTH HAZARDS

The following section describes 1): *EPA's legal authority* to regulate chemicals and compounds that are known or suspected occupational reproductive health hazards, 2) *EPA's activities* concerning reproductive health hazard assessment and management, and 3) an *evaluation* of EPA's activities related to the assessment and management of occupational reproductive health hazards. EPA's authority to address hazards from ionizing radiation are addressed in the section entitled *Nuclear Regulatory Commission*.

Introduction: General Statutory Overview

The statutes that EPA administers do not explicitly address the agency's authority over occupational exposures to known or suspected reproductive health hazards except for the Federal Insecticide, Fungicide, and Rodenticide Act. Under that statute, EPA's mandate includes the protection of farmworkers.[190] In addition, EPA acts under Executive Order No. 10,831 and the Atomic Energy Act to regulate occupational exposure to ionizing radiation, although the agency does not have explicit statutory authority to do so. (This is discussed in a later section.) Despite the lack of an express mandate under the other laws that it administers, however, EPA has considerable authority to acquire and evaluate information concerning reproductive toxicity associated with the production, use, and release of chemicals in the environment. Pursuant to the Toxic Substances Control Act (TSCA), EPA also has extensive discretionary authority to regulate occupational exposures to chemicals in a variety of ways. This authority is presently being evaluated by EPA in relation to several substances, including formaldehyde, glycol ethers, and 1',3' butadiene.

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[190] This mandate was made clear by the 1972 Amendments to the Federal Insecticide, Fungicide, and Rodenticide Act, Pub. L. No. 92-516, 86 Stat. 973 (1972), which expressly addressed the need for farmworker protections.

The following sections discuss the two most important environmental statutes that could be used to regulate or monitor reproductive health hazards from chemical compounds in the workplace: the Toxic Substances Control Act of 1976,[191] and the Federal Insecticide, Fungicide, and Rodenticide Act of 1947.[192] Following this is a description of how particular chemicals that have been associated with reproductive health hazards in the workplace have been dealt with by the current Administration. Five statutes of lesser importance to reproductive health hazards are evaluated in a staff paper available from OTA. These are:

1. the Clean Air Act of 1970, as amended;[192]
2. the Comprehensive Environmental Response, Compensation and Liability Act of 1980 (Superfund);[194]
3. the Solid Waste Disposal Act, as amended by the Resource, Conservation and Recovery Act;[195]
4. the Safe Drinking Water Act;[196] and
5. the Federal Water Pollution Control Act of 1972, as amended by the Clean Water Act of 1977.[199]

Toxic Substances Control Act[198]

TSCA was enacted in 1976 and authorizes EPA to control risks to human health and the environment caused by the production, use, and disposal of toxic substances in the United States. This broad statutory mandate to regulate chemicals throughout their life cycle has provided EPA with a basis for proposing regulatory action affecting several known and suspected reproductive health hazards in the workplace, discussed in detail later.

The term "unreasonable risk" is pivotal to TSCA's implementation. "Unreasonable risk" is not defined anywhere in TSCA despite the fact that the term and its variants are used more than 35 times in the Act. It is clear from various sections of TSCA, however, that EPA's finding of an "unreasonable risk" from a specific chemical substance or mixture will depend, among other things, on the degree of human exposure to the substance, its toxicity and tendency to bioaccumulate in the environment, its use (e.g., as an intermediary or catalyst in the production of a product), and the safety with which it can be disposed. With respect to the weight EPA is to accord each of these characteristics in determining the appropriate regulatory response to a chemical under TSCA, the statute states in § 2(c) that:

> . . . [i]t is the intent of Congress that the Administrator shall carry out this Act in a reasonable and prudent manner, and that *the Administrator shall consider the environmental, economic, and social impact of any action* the Administrator takes or proposes to take under this Act (emphasis added).

This method for assessing risks by weighing other costs is reinforced by the Act's legislative history.[199]

Congress placed extensive discretionary authority in EPA to decide whether or not a public health hazard, regardless of its source or the type of exposure, is better controlled through the use of TSCA than through some other Federal law. It appears that nothing in the language of § 9(a) or its legislative history imposes a barrier to EPA's discretion to decide that a regulatory action under § 6 (regulatory actions) or a § 7 (imminent hazard) order is the best way to protect the public health from significant risks of chemical production, use, or disposal.[200] Section 9(a), however, also allows EPA's Administrator the discretion to conclude that a risk is best prevented or reduced under another Federal law administered by some other Federal agency. This discretionary decision is not subject to judicial review.[201]

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[191] 15 U.S.C. §§ 2601-2629 (1982).

[192] 7 U.S.C. §§ 136-136y (1982).

[193] 42 U.S.C. §§ 7401-7642 (1982).

[194] 42 U.S.C. §§ 9601-9657 (1982).

[195] 42 U.S.C. §§ 6901-6987 (1982).

[196] 42 U.S.C. §§ 300f to 300j-10 (1982).

[197] 33 U.S.C. §§1251-1376 (1982).

[198] 15 U.S.C. §§ 2601-2692 (1982).

[199] See, e.g., S. Rep. No. 698, 94th Cong., 2d sess., reprinted in 1976 U.S. Code Cong. & Ad. News 4491 (stating that "unreasonable" requires a balancing of risks and benefits).

[200] Conf. Rep. No. 1679, 94th Cong., 2d sess., reprinted in 1976 U.S. Code Cong. & Ad. News 4539.

If EPA concludes that another Federal law contains adequate authority to prevent or reduce a suspected or known risk to a sufficient extent, it must submit a report to the other Federal agency and publish it in the Federal Register. This report must describe the risk, including a description of the activity or combination of activities EPA believes presents the risk. It must also request the other agency to determine if the risk may be prevented or sufficiently reduced by action under its authority, as well as whether or not the activity presents an unreasonable risk. TSCA requires that the other agency respond to EPA within 90 days.

If the other Federal agency issues an order declaring that there is no unreasonable risk, or if it initiates a regulatory action, EPA may not take regulatory action under either § 6 or § 7 of TSCA. The Administrator can, however, continue to use his authority under § 4 (Testing), § 5 (Premanufacturing Notification), or § 8 (Reporting and Information Gathering) to insure that more data about the substance (including its production, volume, and use) are collected. Nor does the provision appear to preclude EPA from concluding at some future time that regulatory action is appropriate under TSCA on the basis of new studies. In addition, the Conference Report detailing § 9's mechanisms specified "if the other agency does not take one of these actions (within 90 days) then the Administrator is permitted to act under § 6 or § 7 to protect against the risk."[202]

Section 9(b) attempts to resolve the relationship between TSCA and other environmental laws administered by EPA. It establishes a rule of thumb whereby TSCA is to be used only to the extent that the Administrator determines, in his discretion, that it is in the public interest to use TSCA instead of some other law to regulate the risk. The legislative history of this section reveals that although the determination whether to use TSCA is discretionary, Congress intended the Administrator to make a formal presentation describing why other authorities were not as appropriate as TSCA and why it is in the public interest to resort to TSCA instead of some other act.[203]

Information Gathering

Under TSCA, EPA has numerous ways of developing information about reproductive hazards. Section 4 permits EPA to promulgate *testing rules* prescribing standards for the development of data by the manufacturers of designated chemicals. Section 5 prohibits the manufacture of a new chemical without *prior notification* to EPA, such premanufacture notification (PMN) being accompanied by a minimum set of health and environmental exposure data. Section 8(a) authorizes EPA to require manufacturers to maintain records or submit reports about chemicals not subject to the PMN requirement. Section 8(b) requires EPA to compile and maintain an inventory of chemicals in production and distributed in commerce. Section 8(c) requires chemical manufacturers to maintain records of significant adverse reactions to health or the environment that cause long-lasting or irreversible damage. Section 8(d) directs EPA to promulgate rules requiring chemical manufacturers to submit to EPA copies of health and safety studies conducted by or known to the company. Under § 8(e), a company is required to notify EPA within 15 days of obtaining information that reasonably supports the conclusion that the substance presents a substantial risk of injury to health or the environment. Finally, § 10 requires EPA to carry out research, development, and monitoring whenever necessary to carry out the purposes of TSCA. (These provisions are discussed in detail in Appendix D.1.)

Regulatory Actions[204]

Section 6 allows EPA to select from a broad range of regulatory responses to address significant human health and environmental risks from the production and use of chemicals. The range of possible actions that EPA can take through administrative rules include:

1. prohibiting the manufacture, processing, or distribution of the substance;
2. limiting the amount of such substances that

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[201] Id.

[202] Id.

[203] S. Rep. No. 698, 94th Cong., 2d sess. 11 (1976).

[204] 15 U.S.C. § 2605 (1982).

can be manufactured, processed, or distributed in commerce;

3. prohibiting the manufacture, processing, or distribution of the substance for a particular use;
4. limiting the manufacture, processing, or distribution of a chemical or mixture for a particular use;
5. prohibiting the use of the chemical substance or mixture in a concentration in excess of that specified by the administrator;
6. limiting the concentration of the chemical or mixture in excess of levels specified by the administrator for a particular use;
7. requiring that any such substances be clearly marked with or accompanied by clear and adequate warnings and instructions with respect to their use, distribution in commerce, or disposal, or any combination of such activities (the form and content of labels may be prescribed by EPA);
8. requiring the manufacturer or processor of the substance or mixture to make and retain records of processes used in manufacturing or processing the materials;
9. requiring the manufacturer or processor of regulated substances or mixtures to monitor or conduct tests that are reasonably necessary to assure compliance with any particular rule that EPA has promulgated;
10. prohibiting or otherwise regulating the manner or method of commercial use of the chemical substance or mixture;
11. prohibiting or otherwise regulating the manner or method of disposal of such substance or mixture, or any article containing the material either by the manufacturer or processor themselves, or any persons who use or dispose of such chemical substances or mixtures or articles for commercial purposes; and
12. issuing a directive requiring manufacturers or processors of such substances or mixtures to:
    a. give notice of unreasonable risk of injury to distributors of such materials in commerce, and to the extent that it is reasonably ascertainable, to other persons in possession of or exposed to such substances and mixtures; and to
    b. replace or repurchase such substance or mixture as elected by the person to whom the requirement is directed.[205]

The administrator is also authorized by § 6(a) to limit one or any combination of the above regulatory options to a specified geographic area. (No other environmental statute in the EPA Administrator's arsenal provides this authority.)

Imminent Hazard Authority[206]

Section 7 authorizes EPA to seek orders in the U.S. District Courts to enjoin activities in order to protect against "imminent hazards." Imminent hazards are defined under TSCA as substances or mixtures that present an unreasonable risk of death, serious illness, serious personal injury, or serious environmental harm prior to the completion of an administrative or other proceeding authorized under the bill.[207] In this sense, some reproductive health hazards would fall under the authority of this section.

Public Disclosure of Data[208]

Any information obtained under TSCA that qualifies as a trade secret or as confidential business information generally may not be disclosed to the public, and special clearance is required for employees of the agency who handle this information. However, these data may be disclosed if EPA determines disclosure is necessary to protect health or the environment against unreasonable risk of injury. Regardless of any confidentiality considerations, any information filed pursuant to TSCA's requirements is available to committees of Congress.

Data from health and safety studies are treated separately from the confidentiality protections, however. Pursuant to § 4(b), any health and safety study must be disclosed with respect to any chemical substance or mixture that has been offered for commercial distribution or for which § 4 testing or § 5 notification has been required.[209]

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[205] 15 U.S.C. § 2605(a) (1982).

[206] 15 U.S.C. § 2606 (1982).

[207] 15 U.S.C. § 2606 (1982).

[208] 15 U.S.C. § 2613 (1982).

[209] Section 4(b) adds, however, that disclosure of health and safety studies under TSCA does not authorize the release of any data to the public that disclose processes used in the manufacturing or proc-

Citizen Suit Provisions

TSCA states that any person may petition EPA to issue, amend, or repeal a rule or an order. If the administrator denies or fails to respond to a petition within 90 days, the petitioner may commence a civil action in Federal district court to compel EPA to take the requested action. If the petitioner demonstrates that there is an adequate basis for the issuance of the rule or order requested, the court must order the administrator to initiate proceedings on the requested action,[210] unless doing so would make EPA resources unavailable to attend to more serious problems.[211]

Federal Insecticide, Fungicide, and Rodenticide Act[212]

FIFRA provides a comprehensive mechanism for regulating the use, manufacture, and distribution of pesticides.[213] EPA's authority to regulate reproductive harms from occupational exposures to pesticides under this law is extensive, although not as extensive as it is under TSCA (which confers authority for regulating all uses of chemicals, not just substances used as pesticides). Another reason FIFRA is less potent than TSCA for regulating human health hazards is that the statute is primarily a registration and labeling law. Under limited instances, discussed below, EPA can also suspend and cancel the registration of products classified as pesticides if it determines that the substances are public health hazards.

Congress intended FIFRA to protect the health of farmworkers and other employees exposed to pesticides in the field and in their preparation. In passing the 1972 Amendments to FIFRA, a prime motivation was to make clear EPA's responsibility to protect farmworker health.[214] What is less clear is whether other kinds of workers, including those who dispose of wastes contaminated by pesticides, are similarly protected.

FIFRA's keystone is the registration of pesticide producers and their products. The Act prohibits distributing, selling, or receiving pesticides that are not registered with EPA. In registering a pesticide, EPA can impose restrictions on its use and require labeling to ensure that the pesticide is properly handled and applied. As part of this process, EPA is required to classify pesticides for either general use, restricted use, or a combination of the two. The classification determines who can purchase or apply the pesticide. In general, the law is intended to ensure that the pesticides do not have an "unreasonable adverse effect on the environment." In addition, the statute sets forth procedures for the cancellation and suspen-

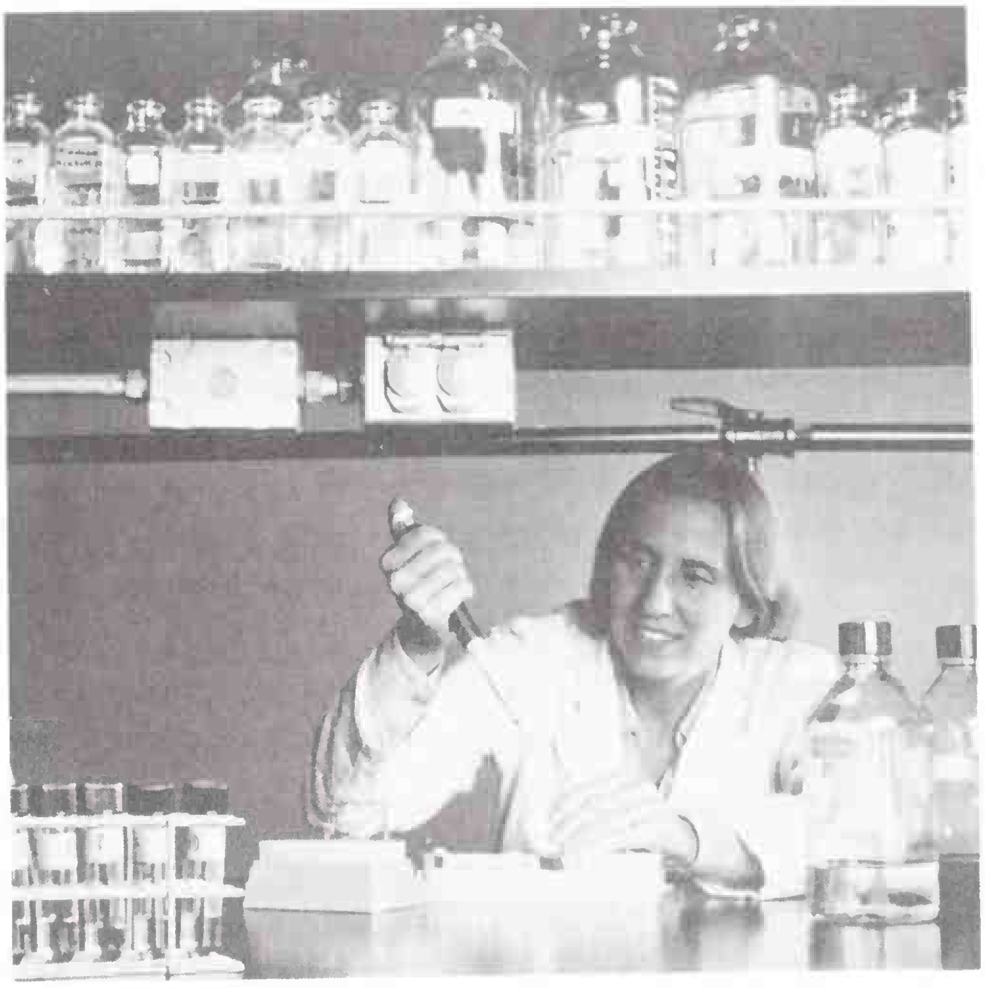

Photo credit: Pemina Meisels

No new chemical may be manufactured unless the manufacturer first provides EPA with exposure and toxicity data.

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essing of chemicals, or the relevant proportions of active ingredients in mixtures. Thus the Administrator must exclude such information when releasing a study. No advance notice to the companies who filed this information is necessary for the release of health and safety studies. To obtain health and safety studies, one must file a Freedom of Information Act (FOIA) request.

[210] See Environmental Law Institute (ELI) Citizen Suit Study for grounds of successful citizen petitions. ELI, Citizen Suits (1984). The most recent action compelled EPA to list formaldehyde under § 4(f) on the basis of animal tests that showed the substance to be a potential carcinogen.

[211] H.R. Rep. No. 1679, 94th Cong., 2d sess. 98 (1976).

[212] 7 U.S.C. §§ 136-136y (1982).

[213] The term "pesticide" refers to any substance or mixture of substances intended for preventing, destroying, repelling, or mitigating any pest, and any substance or mixture intended for use as a plant regulator, defoliant, or desiccant, 7 U.S.C. § 136(u) (definitions). The term embraces a wide variety of biological approaches to the control of pests, including reproductive inhibitors intended to reduce or otherwise alter the reproductive capacity or potential of various organisms and animals.

[214] S. Rep. No. 838, 92d Cong., 2d sess. 4063 (1972).

sion of pesticides that may result in adverse effects on the environment or an imminent hazard.

Two terms—"environment" and "unreasonable adverse effects on the environment"—are pivotal to the use of FIFRA to protect workers from the effects of pesticides. "Environment" includes water, air, land, plants, and humans and other animals, and the interrelationships that exist among these.[215] The phrase "unreasonable adverse effects on the environment" is defined as "any unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits of the use of any pesticide."[216]

Registration of Pesticides[217]

Generally, producers, sellers, and distributors of pesticides must apply for registration of each pesticide with EPA before marketing. The registration process places the burden on the company desiring to market the pesticide to produce the data needed by EPA to evaluate the application.[218] EPA must either approve a registration as expeditiously as possible or deny it according to procedures that give the applicant an opportunity to appeal. For those pesticides the agency decides to register, EPA must classify them for either general or restricted use on the basis of hazards associated with their use.

Regulatory Action on Applications for Registration.—EPA must grant an applicant registration for a pesticide on finding that:

- its composition warrants the proposed claims for it;
- a complete copy of the pesticide's labeling and other material comply with the Act;
- the pesticide will perform its intended function without unreasonable adverse effects on the environment; and
- when used in accordance with widespread and commonly recognized practices, the pesticide will not generally cause unreasonable adverse effects on the environment.[219]

Thus EPA may register a pesticide that has the potential for certain deleterious health effects as long as the risk to man or the environment is not "unreasonable." If EPA determines that use of the pesticide in accordance with its labeling, warnings, and cautions, and in accordance with widespread and commonly recognized practice, will generally not cause unreasonable adverse effects, it can be classified for general use. A pesticide may also be registered for restricted uses if EPA determines that its use may generally cause such unreasonable adverse effects as injury to the applicator unless use is restricted.[220]

Pesticides must be re-registered every 5 years. EPA carries out specific risk/benefit analyses of chemicals suspected of causing unreasonable risks. In addition to the data submissions already described for new pesticides, applicants for re-registration or amendment of an existing registration must also submit to EPA any factual information, including unpublished studies and accident reports, regarding adverse effects of the pesticide on the environment or man that the applicant has obtained or that has come to his attention, and insofar as he is aware, has not previously been submitted to the agency.[221]

Special Review.—If, during the registration of a pesticide or through other information, EPA finds evidence that the pesticide might cause an unreasonable adverse health or environmental risk, § 3(c)(8) authorizes the agency to initiate a "public interim administrative review process" to develop a risk/benefit evaluation for the pesticide. Under this procedure, called "special review," or the "rebuttable presumption against registration" (RPAR) process, the Office of Pesticide Programs develops a recommendation for a regulatory position with regard to the registration, suspension, cancellation, and restrictions on the pesticide under review.

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[215] 7 U.S.C. § 136(j) (1982).

[216] 7 U.S.C. § 136(bb) (1982). The latter phrase appears in several sections of the law. These include a determination by EPA of:
- whether to approve or deny an application for registration of a pesticide,
- whether a pesticide should be classified for general or restricted use,
- whether to issue a notice of intent to cancel registration or to hold hearings,
- whether to suspend a registration pending completion of cancellation procedures,
- whether to issue a final cancellation order, and
- whether a pesticide represents an "imminent hazard."

The relative weight to be assigned to risks and benefits of a pesticide varies with each type of determination, though there is an overriding concern expressed throughout the Act to reduce risk to public health.

[217] 7 U.S.C. § 136a (1982).

[218] FIFRA requires that EPA publish guidelines specifying the kinds of information required to support the registration of a pesticide. 7 U.S.C. § 136a (1982). In cases of minor uses of a pesticide, standards are to be made commensurate with the anticipated extent of use and the level of potential exposure of man and the environment to the pesticide. Furthermore, in the development of these standards, EPA must consider the economic factors of potential volume of use, extent of distribution, and the impact of the cost of meeting the data requirements on the incentives for any potential registrant to undertake the development of the required data.

[219] 7 U.S.C. § 136a(c)(5) (1982).

[220] 7 U.S.C. § 136d(a)(1) (1982).

[221] 40 CFR § 162.8(b)(2) (1984).

Farmworker Protection Standards

Pursuant to EPA's authority to register agricultural pesticides, it is also the primary governmental body with responsibility for overseeing and regulating health risks of these products to farmers and farmworkers.[222] When EPA is exercising this responsibility with respect to a particular class of chemicals, OSHA is preempted from taking action.[223]

EPA first published worker protection standards for agricultural pesticides in May 1974.[224] These rules:

- prohibited applying pesticides when workers who are not wearing protective clothing were in the area being treated,[225]
- prohibited worker reentry until "sprays have dried or dusts have settled," and
- listed harvest intervals for certain pesticides.[226]

In August 1984, EPA published an advanced notice of rulemaking stating that it intended to revise these standards within 12 months.[227] The summary of the notice lists the following areas that EPA intends to consider under its § 3(a) authority, including:

1. expanding the scope of the regulations, including the categories of workers, work activities, and pesticide uses to which the regulations would apply;
2. revising reentry times;
3. revising the protective clothing provisions;
4. revising the standard for warnings; and
5. imposing other types of safety requirements.[228]

EPA also stated that it will consider using new methods to implement and enforce standards.[229]

The current standards give no attention to special subgroups of workers who may be particularly vulnerable to reproductive effects from exposure to pesticides. EPA's 1974 proposal would have defined farmworkers to include children under 12 years of age, who are viewed as being particularly vulnerable to certain types of reproductive health hazards. However, the inclusion of this subpopulation "who might be in the field at any time for any reason" was strongly protested by growers and their associations[230] and therefore dropped as an element in the regulations. In addition, the standards provide no specific precautions with respect to protection for pregnant farm laborers and there is no evidence in the summary of comments received that reproductive harms to pesticide applicators received more than cursory attention.

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[222] S. Rep. No. 838, 92d Cong., 2d sess. 14 (1972). The Senate Committee on Agriculture and Forestry Report on the Amendments stated that EPA's pesticide registration authority encompassed worker protection:

> The Committee believes there can be no question . . . but . . . that the bill (FEPCA) requires the Administrator to require that the labeling and classification of pesticides be such to protect farmers, farm workers, and others coming in contact with pesticides or pesticide residues.

[223] In 1975, the U.S. Court of Appeals for the District of Columbia ruled in Organized Migrants in Community Action v. Brennan that EPA has authority to provide protection for farmers and farmworkers from the adverse effects of pesticides, and that where EPA was exercising this authority, OSHA was preempted from issuing standards on its own. 520 F.2d 1161 (D.C. Cir. 1975). See EPA interpretation at 49 Fed. Reg. 32,605 (1984). This suit arose from citizen suit petitions to compel OSHA to issue permanent standards governing field reentry time for 21 organophosphate pesticides for which OSHA had issued emergency temporary standards in 1973. 38 Fed. Reg. 10,715 (1973). Publication of emergency standards under OSHA started the 6-month period within which the agency must issue final standards. However, during this period, EPA had indicated its intention to publish standards and had signed a memorandum of agreement with OSHA to this effect. 39 Fed. Reg. 9457 (1974). The Court held on this basis that OSHA was preempted from taking action with regard to this class of chemicals by EPA's action.

[224] 39 Fed. Reg. 16,888 (1974) (codified at 40 CFR pt. 170 (1982)). The 1974 standards define "farmworker" or "worker" as "any person or persons engaged in agricultural hand labor in the field." 39 Fed. Reg. at 16,890; 40 CFR § 170.2(b). This term encompasses workers who might come in contact with pesticides during transportation, storage, application, or after the product has been applied.

[225] 39 Fed. Reg. at 16,890; 40 CFR § 170.2(c).

[226] "Harvest intervals" or "reentry" times were set for 12 substances which precluded unprotected workers from reentering a field treated with pesticides for specified periods ranging between 24 and 48 hours.

[227] 49 Fed. Reg. 32,605 (1984); 40 CFR § 170.3(b)(2) (1984) (intervals specified). Farmworker organizations and some EPA officials believe that the 1974 standards provide inadequate protection for field laborers, particularly against reproductive hazards and other health concerns. See 49 Fed. Reg. 32,605 (1984).

[228] 49 Fed. Reg. at 32,605.

[229] Id. at 32,605, 32,608. EPA's enforcement authority is presently limited to instances when it can show that a product has been used inconsistently with its labeling.

[230] See 39 Fed. Reg. 889 (1974).

Use of Restricted Pesticides[231]

Section 4 authorizes EPA to prescribe standards for the certification of applicators of pesticides subject to restricted use under § 3. By means of this provision, EPA can minimize exposure to designated toxicants, including substances that may be reproductive health hazards, by requiring that persons who mix and apply the substances be certified. A certified applicator must demonstrate practical knowledge of application techniques, environmental factors, and pesticide toxicity, through written examinations and in some cases performance testing.[232]

Cancellation and Reregistration of Pesticides[233]

The provisions of § 6 may be directly relevant to the detection and removal of pesticides from the market that may expose workers to possible reproductive health hazards. Section 6(a) requires EPA to automatically cancel a pesticide's registration after 5 years unless a request for continuance of the registration is submitted and approved. Section 6(b) authorizes EPA to cancel pesticides that cause unreasonable adverse effects on the environment or man. Finally, § 6 provides EPA with authority to suspend the registration of a pesticide immediately to prevent an "imminent hazard." (These provisions are discussed in Appendix D.2.)

Storage, Packaging, and Disposal of Pesticides[234]

Section 19 of FIFRA authorizes EPA to establish procedures and regulations for the safe storage, packaging, and disposal of pesticides. EPA must accept for disposal, on request of the owner, any pesticides for which registration has been been canceled. General precautions for the handling of pesticide wastes have been promulgated by EPA.[235] Chemicals associated with reproductive health hazards do not appear to be handled differently than other toxic wastes.

EPA Implementation of Reproductive Health Hazard Control Programs

The foregoing discussion indicates that EPA has clear authority under both TSCA and FIFRA to regulate certain types of occupational exposures to reproductive health hazards and to collect information about the potential reproductive effects of various substances as a basis for regulatory action. It is also clear that under a wide variety of other statutory programs (see staff paper available from OTA), the agency may accumulate data and assess a substance's potential for developmental health effects, mutagenicity, and other reproductive impacts associated with human and environmental exposure in the three environmental media. The following sections present an overview of what EPA has done in the area of reproductive hazard assessment and management. This information was primarily developed from discussions with EPA staff members. Finally, relevant interagency relationships, particularly between EPA and the Occupational Safety and Health Administration, the Food and Drug Administration, and the Consumer Protection and Safety Commission are described.

As was discussed earlier, EPA has statutory authority to regulate chemicals on the basis of developmental effects, as well as on the basis of other more subtle reproductive and sexual impacts. EPA receives and analyzes test data of these health effects under TSCA and FIFRA, and routinely performs risk assessments based on these characteristics. Although it appears that carcinogenic characteristics of a chemical generally provide a more compelling basis for regulation by EPA than do reproductive health effects, this emphasis may change, particularly with the development and acceptance of short-term tests. According to several EPA officials, EPA regulates chemicals on the basis of carcinogenicity more often than for reproductive effects because of the assumption that chemicals that cause reproductive health effects generally also have positive indicators of carcinogenicity. The problem with reproductive health hazards, as has been pointed out in congressional testimony,[236] is that regulation on the basis of carcinogenicity is generally inadequate to protect against the deleterious reproductive effects that may occur at lower dosages.

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[231] 7 U.S.C. § 136(b) (1982).

[232] 40 CFR § 171.4 (1984).

[233] 7 U.S.C. § 136d (1982).

[234] 7 U.S.C. § 136q (1982).

[235] 40 CFR pt. 165 (1984).

There are, however, some prominent examples of EPA actions taken on the basis of reproductive health effects alone. The regulatory activity surrounding several of these chemicals where occupational exposure was involved is discussed below. It should be noted that all of the final actions based on reproductive health effects have occurred pursuant to FIFRA. Several important actions involving occupational exposures to chemicals under TSCA are also pending in EPA.

EPA Actions Under FIFRA

Dibromochloropropane (DBCP).—[See discussion of EPA and OSHA regulation of this nematocide in the section entitled *OSHA Reproductive Health Hazard Regulations.*]

Ethylene Oxide (EtO).—[See discussion of EPA and OSHA regulation in the section entitled *OSHA Reproductive Health Hazard Regulations.]*

Oryzalin.—In November 1979, the International Chemical Hazards Union petitioned EPA under FIFRA to ban the production and use of the herbicide Oryzalin based on anecdotal evidence of high rates of birth defects in the offspring of workers involved in the production of Oryzalin at a plant in upstate New York. During a 1½-year period of the pesticide's production, not one of the worker's wives had experienced a normal pregnancy.[237]

In March 1980, EPA decided not to regulate Oryzalin, based on its review of a series of developmental studies performed by Eli Lilly, whose subsidiary produced Oryzalin. Analysis of eight other plants involved in the production of the pesticide showed no statistically significant rate of birth defects. Although one developmental test on laboratory rabbits produced evidence of teratogenesis,[238] replication of the test produced no effect, and EPA judged it to be an insufficient basis for regulation.[239] The agency concluded that production methods at the upstate New York plant were less protective than in other plants, allowing greater exposure to the chemical.[240] EPA officials were denied entrance into the plant to test this hypothesis, because they did not have legal authority for inspections of working places under either TSCA or FIFRA.[241] While the agency agreed to do further monitoring of Oryzalin (along with OSHA and NIOSH), EPA officials concluded at that time that they did not have the authority to regulate the production of pesticides, only their use.[242]

Cyanazine.—EPA has recently undertaken a special review of cyanazine, a herbicide marketed under the trade name of Bladex,[243] after the agency found that cyanazine causes developmental effects in laboratory animals. As a result of these studies, EPA has concluded that female agricultural workers who apply, load, or mix the herbicide may be exposed to unsafe levels of the substance.

EPA determined that a dietary risk of adverse effects of cyanazine as a result of traces found in agricultural products was insignificant. The agency is currently undertaking an analysis of potential adverse effects of the herbicide on drinking water, however.

Because of the effects on laboratory animals, the agency has required that warning labels be placed on the herbicide notifying users of these potential effects. Furthermore, because of the possibility of ground or surface water contamination, labels must be placed on cyanazine advising applicators not to use the substance in permeable soils or where water is near the surface.

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[236] Relationship of Exposure to Toxic Chemicals to Reproductive Impairment, Hearings Before the Subcommittee on Investigations and Oversight of the House Committee on Science and Technology, 97th Cong., 2d sess. 25-26 (1982) (statement of Dr. Jeane Manson, University of Cincinnati Medical School).

[237] See Relationship of Exposure to Toxic Chemicals to Reproductive Impairment, Hearings Before the Subcommittee on Investigations and Oversight of the House Committee on Science and Technology, 97th Cong., 2d sess. 122-128, 142-145 (1982) (statements of Dr. Gordon F. Hueter, Director, Health Effects Laboratory, and Dr. Ed Johnson, Director, Office of Pesticides, EPA).

[238] Id. at 123.

[239] Id.

[240] Id. at 126.

[241] Id.

[242] Id.

[243] 50 Fed. Reg. 14,151 (1985).

During the special review of cyanazine, EPA will receive evidence and determine what final action to take, including whether to issue a final notice to propose regulations to reduce the risks associated with cyanazine or issue a notice of an intent to cancel the herbicide.[244]

Nitrophen (TOK).—Regulation of the herbicide nitrophen, marketed under the trade name of TOK, was considered by EPA based almost solely on the teratogenic risks to female farmworkers.[245] In 1980, however, the company that produced the compound voluntarily withdrew it from the market. The company intended to develop safe uses for the chemical and return it to the market, but laboratory tests performed by both the agency and the company could find no level at which the compound did not have a teratogenic effect. In 1983, EPA requested that the company proceed with cancellation of the product, "in light of the determination that nitrophen presents a substantial teratogenic risk and a potential oncogenic and mutagenic risk without economic benefits."[246] The company agreed to the cancellation, and EPA completed cancellation proceedings in 1984.

Agency Actions Under TSCA

The Glycol Ethers.—EPA published an Advanced Notice of Proposed Rulemaking (ANPR) to regulate two glycol ethers and their acetates in the Federal Register in January 1984 pursuant to its authority in § 6 of TSCA.[247] The notice stated:

> A number of animal studies indicate that adverse reproductive and fetotoxic effects are associated with these chemical substances at concentrations to which humans may be exposed. EPA is concerned about both short-term and chronic exposure of pregnant women, either as workers or as household consumers, to these chemical substances. EPA is also concerned about the exposure of males to these substances, both from short-term and chronic exposure. EPA has also made a preliminary review of the toxicity of some potential substitutes for these four ethers, and while some exhibit toxic effects, they appear to be of less concern than the effects of the glycol ethers that are the subject of this ANPR.[248]

According to an EPA official, this is the first regulatory action under TSCA based solely on reproductive health hazards.[249] The Advanced Notice of Proposed Rulemaking followed earlier reports that the Office of Toxic Substances had been "actively pursuing regulation" of six chemicals, including glycol ethers, that are used as intermediaries in the production of plastics.[250] These same reports also indicated that EPA had been attempting to coordinate regulation of glycol ethers with the Consumer Product Safety Commission (CPSC) and OSHA.[251] However, CPSC rejected the notion of a coordinated effort with EPA and had determined earlier to take no action on the group of compounds used commonly as solvents in household products and paints.[252] As a result, publication of the notice was a unilateral action by EPA, and does not refer to cooperative regulation of glycol ethers with other agencies.

This use of TSCA to control glycol ethers based on their potential reproductive effects in the workplace, though still at a pre-regulatory phase, has provoked some controversy within the agency.[253] The Reproductive Effects Assessment Group (REAG) refused to approve the risk assessment performed by the Office of Toxic Substances when it came to the office for review because it employed what REAG considered to be questionable uses of dose-response relationships in its risk assessment.[254] Despite these conflicts, officials from both offices believe that EPA will undoubtedly regulate glycol ethers based on their reproductive effects. A partial ban on some uses is apparently being considered.

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[244] Id.

[245] Personal communication with Harry Chitik, Office of Pesticide Programs, EPA (Sept. 8, 1984).

[246] Chem. Reg. Rptr. (BNA) 141 (May 4, 1984).

[247] 49 Fed. Reg. 2921 (1984).

[248] Id.

[249] Personal communication with Harry Teitelbaum, Office of Toxic Substances, EPA (Sept. 20, 1984).

[250] Chem. Reg. Rptr. (BNA) 1301 (Jan. 1, 1983).

[251] Id.

[252] Personal communication with Harry Teitelbaum, Office of Toxic Substances, EPA (Sept. 20, 1984).

[253] Personal communication with Peter Voytec, Reproductive Effects Assessment Group, Office of Research and Development, EPA (Sept. 20, 1984).

[254] Personal communication with Harry Teitelbaum, Office of Toxic Substances, EPA (Sept. 20, 1984).

Industry opposes EPA's regulation of glycol ethers, claiming that the agency lacks sufficient data on these chemicals' uses, exposures, benefits, and suspected risks.[255] In addition, industry representatives believe that EPA should defer regulation of glycol ethers to OSHA because that agency is responsible for regulating workplace hazards.[256]

Other Actions

A summary of EPA actions under TSCA and FIFRA based on information from EPA's February 1984 Status Report of Chemical Activities appears in table 7-3. It shows the number of chemicals that EPA has looked at or is looking into under the authority of the two acts based on mutagenic, developmental, and reproductive effects, single or in any combination. Listing of these chemicals based on any of these effects in EPA's data base does not necessarily preclude their listing in another category of effects, such as carcinogenicity. Therefore, reproductive effects may not be the sole basis for the EPA actions described.

Interagency Jurisdictional Issues

EPA's activities concerning reproductive health hazards to workers, as illustrated by the ethylene oxide and glycol ethers cases, suggest a growing tension between EPA and OSHA on jurisdictional issues. EPA's increased willingness in the past several years to rely on the use of TSCA, with its very broad mandates to regulate not only the initial manufacture of chemical substances, but also their use and disposal, has created a potentially volatile situation between the two agencies.

Table 7-3.—EPA Actions Under TSCA and FIFRA Based on Mutagenicity, Developmental, and Reproductive Effects[a]

TSCA		FIFRA	
Testing	250		
Preliminary/preregulatory	120	Preliminary/regulatory assessment	2
Summary review	18	Cancellation/ban	14
Ban	0	Other/DCI	42
Notice	5	Risk documentation/assessment	6
Special review	96		

[a]Four substances listed under both Acts are not included.

SOURCE: Adapted by Environmental Law Institute from information provided by the U.S. Environmental Protection Agency.

In an effort to resolve some of the more outstanding political issues that EPA's actions over recent months have created, EPA and OSHA are considering a comprehensive Memorandum of Understanding for controlling workplace exposures giving EPA broad discretion as to whether or not it will refer chemicals to OSHA. Until this Memorandum is formalized, EPA has completed an intra-agency memo outlining interim policy for referring actions to OSHA and other agencies. The document states that EPA will use TSCA § 9(a) to refer a chemical problem to OSHA as soon as: 1) there is credible evidence that the chemical poses an unreasonable risk, and 2) EPA has reason to believe that the problem would be most effectively or efficiently addressed under the provisions of the OSH Act, or the Mine Safety and Health Act (MSH Act). It also states that referral will be made where occupational exposures are at issue, or where the exposure could be most effectively addressed by workplace standards. These statements are simply a reiteration of TSCA's language. According to the memo, however, EPA will not refer a chemical to OSHA when "too much of the exposure lies beyond the reach of the OSH Act and MSH Act" and where "a full or partial ban on the production or use of the chemical, or other remedies uniquely available under § 6 of the TSCA, provide the most effective or efficient remedy."

This approach has been criticized by industry groups and by OMB, both of whom claim that § 9(a) of TSCA should not be used to preclude OSHA from exercising its authority over workplace exposures. However, a letter from three members of the Senate Environment and Public Works Committee endorsed this approach, saying that the provision "does not preclude action under TSCA merely because another agency also has the authority to respond."[257]

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[255] Chem. Reg. Rptr. (BNA) 5 (Apr. 6, 1984).

[256] Personal communication with Sanford Gaines, Assistant General Counsel, Chemical Manufacturers Association (Oct. 27, 1984).

[257] Chem. Reg. Rptr. (BNA) 276-277 (June 1, 1984).

Qualitative Analysis of EPA Management of Reproductive Health Hazards

Many of the EPA officials interviewed for this report stated that there had been very little activity within EPA to regulate chemicals with reproductive effects. Some felt that EPA has become more seriously involved in this area, but that this was a fairly recent development. Only a few individuals were knowledgeable about EPA's efforts to use its existing authority under environmental statutes to examine occupational exposures to chemicals with known or suspected reproductive effects.

There was virtual agreement among interviewees that EPA tends to look first at chemicals based on their potential to cause cancer. They believed this to be largely a result of statutory authority, congressional pressure, and public phobia about carcinogenic chemicals. There was disagreement among interviewees, however, concerning whether regulating a particular chemical based on its carcinogenic risk provided sufficient protection to people from the reproductive health hazards of some substances. (A discussion of EPA risk assessment activities appears in chapter 6.)

Many public interest groups and some government officials expressed reservations about EPA's willingness to use its authority to protect workers. Some charged that EPA's inactivity in this regulatory area was due to the agency's lack of a mission to protect public health in the process of regulating chemicals. Many others, however, expressed doubts about EPA's authority to regulate occupational exposures and its leaders' willing-

Box 7C.—OMB's Role in the Regulatory Process

The Office of Management and Budget (OMB) has assumed steadily greater responsibility for regulatory affairs since its establishment in 1970. An outgrowth of the President's Bureau of the Budget, OMB was first given a significantly larger role in regulatory oversight by President Nixon, who issued an internal directive requiring OMB to review agency rules. Thereafter, Presidents Ford and Carter both took additional steps to bring agency rulemaking under centralized presidential control. President Ford issued Executive Order 11,821 [258] requiring an "inflation impact statement" to accompany regulations with a major economic impact. During President Carter's tenure, Executive Order 12,044 [259] was issued, providing for the preparation of a government-wide agenda on upcoming rules and regulatory analyses on major regulations, as well as establishing an ad hoc "Regulatory Analysis Review Group" to review certain major rules. By the time President Reagan took office, OMB had been given additional authority to control information requests issued by Federal agencies under the 1980 Paperwork Reduction Act.[260] President Reagan further increased OMB's role by setting up an OMB-staffed Task Force on Regulatory Relief and by issuing Executive Order 12,291.[261] The most significant components of the Executive Order authorized OMB to:

1. determine whether agency rules are "major," triggering a requirement for the agency to write a regulatory impact analysis (RIA); and
2. ensure that, "to the extent permitted by law," agencies issued regulations only if "the potential benefits to society for the regulation outweigh the potential costs to society."

The enactment of the 1981 Regulatory Flexibility Act,[262] administered by OMB and designed to protect small businesses from over-regulation, further consolidated OMB's role in the regulatory process.

The latest presidential decree, Executive Order 12,498,[263] established a new regulatory planning process in which EPA, OSHA, and other executive agencies will be required to submit annual draft regulatory programs to OMB. The new process means that OMB will be involved in rulemaking much earlier and much more broadly than in the past, observers say.[264] OMB currently plays a critical role in nearly every aspect of regulatory affairs and executive budget decisions.

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[258] Exec. Order No. 11,821, 3 CFR § 1180 (1974).

[259] Exec. Order No. 12,044, 3 CFR § 152 (1979).

[260] 44 U.S.C. § 3501 (1982).

[261] Exec. Order No. 12,291, 3 CFR § 127 (1982).

[262] 5 U.S.C. §§ 601-602 (1982).

[263] Exec. Order No. 12,498, 50 Fed. Reg. 1036 (1985).

[264] OMB's Rule-Making Role Could Change Legal Process, Nat'l. L.J., Jan. 28, 1985 at 8, col. 4.

ness to insert themselves into this politically "hot" area, given OSHA's current reticence on the issue. Several officials and other interviewees expressed high regard for EPA's current emphasis regarding several proposed actions under TSCA and FIFRA to regulate significant occupational and consumer reproductive health hazards.

Inter- and Intra-agency Coordination

Interviewees inside and outside the agency noted that the management of reproductive health hazards in the workplace could benefit from improved inter- and intra-agency coordination. EPA officials noted that there was little formal coordination between other program offices and REAG with respect to how chemicals associated with reproductive effects are evaluated. A workgroup on teratology has recently been established in the agency, but beyond this group, communication with other officials is on an informal basis. There was also a lack of formal communications with other agencies. This may be remedied by a newly organized Intra-agency Risk Management Council now under the Cabinet Council and its subcommittee on reproductive health hazards, but few interviewed thought there was real hope for this forum providing meaningful communication channels among agencies. In addition, some thought that a formal Memorandum of Understanding between EPA and OSHA would probably not cure strained relationships between the agencies due to the use of TSCA for regulating occupational hazards.

Future of Reproductive Health Hazard Program at EPA

Several interviewees suggested that EPA had essentially failed to regulate reproductive health hazards to farmworkers despite a strong statutory mandate under FIFRA and that EPA is generally unresponsive to the special working conditions of farmworkers, who may be exposed to greater quantities of toxic substances than any other work force in the country as a result of "spray drift" and lack of clean drinking water. Since many farmworkers do not have laundry facilities, they often wear pesticide-laden clothing for days at a time, including in their homes. Most farmworkers do not have drinking water facilities in the field, so they rely on irrigation ditches as a source of water. These ditches are commonly used to transport a mixture of water and pesticides. While no studies have directly determined the causes of reproductive difficulties some farmworkers are experiencing, several interviewees claim there is a "high index of suspicion" relating it to pesticides in drinking water. None of these individuals was optimistic that EPA's current attempt to address some of these problems by revising worker standards will be successful.

Many people who are encouraged by EPA's interest in reproductive health hazards from chemicals are generally not optimistic about whether this interest can provide a solid foundation for regulating chemicals on the basis of their potential to cause deleterious reproductive effects. Many believe that the basic science in this area is seriously deficient. Lacking a sufficient scientific data basis, the proposed risk assessment guidelines, one person stated, may be putting the cart in front of the horse.

Another related theme that emerged during the interviews was curiosity, and general despondency, about the future of these programs under new EPA leadership. The importance of the publication of the reproductive risk assessment guidelines for public comment before January 1985 was stressed by several people. Interviewees seemed to believe that former Administrator Ruckelshaus' leadership was fundamentally responsible for placing emphasis on reproductive effects as an issue and in the agency's present willingness to challenge OSHA's jurisdiction in this area.

Conclusions

The Environmental Protection Agency has made significant strides within the last several years toward developing its institutional expertise and authority for regulating occupational exposures on their potential to induce deleterious reproductive effects. However, it is also apparent that while the statutory authority for regulating these health risks undeniably exists under the Toxic Substances Control Act, and to a more limited extent under the Federal Insecticide, Fungicide and Rodenticide Act, there are some substantial scientific, institutional, and political uncertainties that may militate against EPA assuming a larger role than it now has in regulating occupational reproductive health hazards.

One of the most important problems confronting EPA (and any other agency) in regulating reproductive health hazards appears to be scientific. The "state-of-the-art" for assessing hazards or risks for different types of reproductive effects is only beginning to evolve.

There are also institutional constraints on EPA's ability to regulate reproductive health hazards effectively. First, it is not clear whether EPA's collection of data on reproductive health hazards is sufficiently systematized to provide a regular and consistent data base for assessing chemicals across the board for their reproductive effects. The new FIFRA regulations will, for the first time, require manufacturers and processers of pesticides already registered by EPA to submit information on these products' potential for reproductive effects. In addition, information collected on the reproductive health effects of new and existing chemical compounds under TSCA may not be uniformly available to other program offices, including the Office of Pesticide Programs. The agency may also be legally prohibited from sharing this information with OSHA, except in certain instances such as TSCA § 9 referrals. Finally, there seems to be a notable dependence on EPA's part to rely on informal relationships between professionals within the agency and with health professionals in the private sector to stay abreast of current university studies and publications on the reproductive effects of chemicals and scientific assaying techniques. These communication channels are based, at least in part, on EPA employees' membership in scientific societies as well as former professional and collegial associations. These techniques, while consistently important in scientific communities in private institutions as well as in the government, are sufficiently personal in nature that they may not necessarily become part of the institutional memory of the agency when important staff professionals leave EPA.

The third largest area of concern is political constraints on EPA's ability to regulate occupational health hazards in general, and reproductive health hazards in particular. Although EPA has moved to regulate such chemicals as ethylene oxide, formaldehyde, and glycol ethers, all of which may have potential reproductive effects in humans but that are nonetheless used widely in the workplace, there is a perception among the EPA staff working on these actions that this is the result of EPA's recent leaders' willingness to use TSCA and FIFRA to take the initiative to manage these hazards. The memorandum outlining EPA's position on the future Memorandum of Understanding to be consummated between EPA and OSHA concerning EPA's authority to use TSCA for occupational exposures, for example, demonstrates very little willingness by EPA to yield its jurisdiction over these hazards to OSHA. In the situation involving EPA's proposal to regulate ethylene oxide use in hospitals on the basis that the compound was registered under FIFRA as a pesticide, the same aggressiveness appears evident. According to interviewees, the agency relented only when convinced by public interest groups of the importance of letting OSHA proceed in setting workplace exposures so as not to run afoul of the holding, in *Organized Migrants in Community Action* v. *Brennan*,[265] that EPA's actions could preempt OSHA if it moved to regulate the chemical even though EPA did not have the clear authority or resources to inspect or enforce EPA regulations.

Yet, EPA has indicated that it will refer two other chemicals, 4',4' methyene dianiline and 1'3 butadiene, over which EPA and OSHA share potential jurisdiction, to OSHA under § 9 of TSCA, since it believes OSHA can most effectively regulate human exposures to these chemicals. EPA has not yet formally referred these chemicals. The agency is currently preparing regulatory packages for referring methyene dianiline and 1'3 butadiene to OSHA as well.

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[265] 520 F.2d 1161 (D.C. Cir. 1975).

NUCLEAR REGULATORY COMMISSION (NRC)

Despite early awareness of the hazards of occupational exposure to radiation,[266] a Federal regulatory response was belated. The development of nuclear technology during World War II and dramatic demonstration of its biological destructiveness did not immediately elicit a Federal response to protect health. Rather, the Atomic Energy Act of 1946[267] showed congressional preoccupation with maintaining both secrecy and the Government's monopoly on nuclear technology. The Act made no substantive statement on public or occupational health.[268]

Congress modified this course with the Atomic Energy Act of 1954.[269] Intent on finding peaceful uses of atomic energy, Congress encouraged private participation in the development of nuclear technology. The result was a substantial growth in the use of radioactive materials in industry and Government coupled with increasing use of X-rays and radioisotopes in medicine, leading to a corresponding increase in the size of the work force exposed. In 1960, crude estimates indicated that approximately 440,000 workers were exposed. By 1970, the number had grown to an estimated 775,000, an increase of 80 percent in 10 years.[270] By 1980, about 1.3 million workers were being exposed to radiation. Of these, 44 percent were exposed in medicine, 23 percent in industries not part of the nuclear fuel cycle, 16 percent in Government, 11 percent in the nuclear fuel cycle, and 6 percent in miscellaneous occupations.[271]

Congress had anticipated this trend; the 1954 Act represented the first substantive Federal involvement in protecting the health of workers exposed to radiation. Under this Act, the Atomic Energy Commission (AEC) was charged with the duty to enact regulations to protect health,[272] and in 1957, it issued its first Standards for Protection Against Radiation.[273] In 1959, the Federal Radiation Council was established to advise the President on radiation matters affecting health, and in 1960, it promulgated the first Federal Radiation Guidance for occupational exposure to radiation.[274] The Council was abolished in 1970, when the Environmental Protection Agency was created, and its functions were transferred to the new agency.

Today, no single agency regulates radiation exposure of workers; Federal responsibility, which is dispersed among five executive departments, one independent commission, and two agencies, by diverse statutory provisions, operates under the unifying force of Federal radiation protection guidance administered by EPA. However, by 1980, a major review had found "inconsistencies of jurisdiction and regulatory programs. . ." and "confusion . . . from inconsistencies in ways in which regulatory agencies and the public regard and interpret data . . . [and] what the policy should be."[275] The primary authority for the regulation of occupational exposure to ionizing radiation in the nuclear industry rests with the Nuclear Regulatory Commission (NRC), the successor agency to AEC. In the medical and industrial communities, EPA's authority is shared with OSHA and the States.

The Energy Reorganization Act of 1974 created NRC and abolished AEC.[276] AEC had been given the sometimes conflicting roles of both promoting and regulating nuclear technology. The reorganization established NRC as an independent commission that inherited only AEC's regulatory responsibilities.[277]

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[266] See, e.g., historical review in D. Serwer, The Rise of Radiation Protection, 1896-1935, Report to Brookhaven National Laboratory, BNL, 22,279 (December 1976).

[267] 42 U.S.C. §§ 2011-2296 (1982).

[268] W. Wood, Nuclear Safety: Risks and Regulation, American Enterprise Institute, (1983).

[269] 42 U.S.C. §§ 2011-2296 (1982).

[270] A.W. Klement et al., Estimates of Ionizing Radiation Doses in the U.S.: 1960-2000, EPA, ORP/CSD 72-1 (1972).

[271] S. Kumazawa, D. Nelson & A.C.B. Richardson, Occupational Exposure to Ionizing Radiation in the United States: A Comprehensive Summary for the Year 1980 and a Summary of Trends for the Years 1960-1985, Office of Radiation Programs, EPA (1984). See also L. Sagan, Radiation and Human Health, vol. 4, No. 7 EPRI J. (September 1979).

[272] 42 U.S.C. § 2201(b) (1982).

[273] 10 CFR pt. 20 (1958).

[274] 25 Fed. Reg. 4402 (1960).

[275] Report of the Task Force on Occupational Radiation Exposure Regulations, U.S. Radiation Policy Council (1980).

[276] 42 U.S.C. §§ 5801-5891 (1982).

[277] See discussion in W.C. Wood, Nuclear Safety: Risks and Regulation, American Enterprise Institute (1983). See also 10 CFR pt. 1 (1984).

NRC's authority is conferred by three statutes: the Atomic Energy Act of 1954,[278] the Energy Reorganization Act of 1974,[279] and the Uranium Mill Tailings Radiation Control Act of 1978.[280] The Commission's regulatory power is derived principally from the authority previously held by AEC, since all licensing and rulemaking functions of AEC conferred by the Atomic Energy Act were transferred to NRC by the Energy Reorganization Act. As a result, NRC's jurisdiction over human exposure to radiological hazards pertains to exposures to "source, byproduct and special nuclear material."[281] NRC's regulatory jurisdiction runs with all materials included in these categories. However, NRC authority is limited to NRC-licensed activities. Furthermore, NRC's regulations are subject to EPA environmental radiation protection standards for air and water, which have been established for all components of the nuclear power cycle and for all emissions to air from any other licensed operation.

If a material is reactor-produced (e.g., Americium used in smoke detectors) or is a source material (e.g., uranium used in ceramic dyes or thorium used in welding rods), then NRC may regulate the workplace in which it is used, under some circumstances. Thus, NRC with its comprehensive control over nuclear power plants, also finds smoke detector and ceramic manufacturing plants within its jurisdiction.[282] Regulated workplaces may include nuclear power reactors; reactor fuel producers; uranium milling; and all industrial, manufacturing, medical and pharmaceutical facilities that use controlled materials.

The Uranium Mill Tailing Radiation Control Act[283] extended NRC's authority by expanding the definition of "byproduct material" to include uranium and thorium mill tailings. Congress recognized the radiological hazard posed by tailings and directed NRC to subject this class of byproduct to appropriate regulatory control, under standards established by EPA, to protect health and the environment.

NRC implements its statutory authority in three main ways: licensing proceedings, rulemaking, and regulatory guides. NRC also has the authority to relinquish some of its regulatory power to State radiation control programs (Agreement States). In addition, States may establish standards, applicable to all NRC licensees, that are more restrictive than those set by EPA under the Clean Air Act.

Important elements of nuclear safety regulation have developed through NRC licensing proceedings. NRC has authority to regulate by license most aspects of nuclear technology. Atomic Energy Act materials are therefore licensed on a cradle-to-grave basis; licenses are necessary to distribute, possess, use, transport, and dispose of nuclear material. Nuclear production and utilization facilities also undergo extensive licensing procedures in two steps: at the construction permit stage and at the operating permit stage. The NRC staff reviews safety aspects at each stage. At the end of the process, a license may be issued with whatever restrictions are determined necessary for the safe operation of the plant. Throughout the process, there is a strong presumption that the facility can be made acceptably safe; NRC has never denied an operating license to a constructed nuclear facility.[284] In all licensing proceedings, NRC establishes minimum criteria requisite to the issuance of a license,[285] and can condition the license on terms that force the licensee to comply with all NRC rules, regulations, and orders.[286]

NRC also has broad authority to promulgate *regulations* that govern licensee activities, and many regulations have been adopted by NRC to resolve safety and occupational exposure issues on a generic basis, applicable to all licensees.[287]

Regulatory guides are also issued by NRC to describe acceptable methods of compliance with NRC regulations. While not legally binding, the expense for the licensee of demonstrating alternative means of compliance makes acceptance of the NRC methods practical. The guidelines are so detailed that licensees often have little leeway for developing alternative methods of promoting safety.[288]

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[278] 42 U.S.C. §§ 2011-2296 (1982).

[279] 42 U.S.C. §§ 5801-5891 (1982).

[280] 42 U.S.C. §§ 7901-7942 (1982).

[281] 42 U.S.C. § 2014 (1982).

[282] Senate Committee on Goverment Affairs, Study on Federal Regulation: Regulatory Organization, S. Doc. No. 91, 95th Cong., 2d sess. (1977).

[283] 42 U.S.C. §§ 7901-7942 (1982).

[284] W.C. Wood, Nuclear Safety: Risks and Regulation, American Enterprise Institute (1983).

[285] 42 U.S.C. §§ 2073, 2093, 2111 (1984).

[286] 42 U.S.C. §§ 2201(b), 2233 (1984).

[287] See 10 CFR pts. 19, 20 (1984).

NRC has the authority to relinquish specific regulatory powers to a State by written agreement, but it may not delegate its responsibility for special nuclear materials in quantities sufficient to form a critical mass,[289] for the production or operation of nuclear facilities,[290] for the export or import of nuclear materials or facilities,[291] or for certain disposal methods of nuclear materials.[292] Before entering an agreement with a State, the Commission must determine that the State radiation protection program is sufficiently compatible with that of the Commission.[293]

All licensees are governed by NRC's occupational exposure regulations contained in 10 CFR Parts 19 and 20. Since NRC Agreement States must have compatible regulations, these States effectively implement the 10 CFR Parts 19 and 20 regulations. In 1983, NRC had agreements with 26 States, which had issued about 13,200 radioactive material licenses. This represented approximately 64 percent of all licenses issued in the United States.[294]

Few NRC actions relevant to radiation and reproductive health have been tested by judicial review. (A discussion of those actions that have been reviewed appears in a staff paper available from OTA.)

Other Regulatory Authority

Several other agencies have statutory authority to set and enforce standards for worker exposure to radiation. The most important of these is the overall Federal guidance provided by the Environmental Protection Agency. In 1970, EPA was directed by *Reorganization Plan Number 3* to assume the functions of the former Federal Radiation Council to "advise the President with respect to radiation matters, directly or indirectly affecting health, including guidance for all Federal agencies in the formulation of radiation standards. . . ."[295] Under this authority, EPA studies the hazards of exposure to radiation and formulates guidance for use by other agencies.[296] All Federal regulations are consistent with this guidance.[297] In the case of occupational exposure, this guidance includes numerical limits on the exposure of workers. This guidance, which was last issued in 1960, has recently been reviewed and new recommendations are in the final stage of review by Federal agencies.

Although NRC and the States are not bound by EPA guidance, they have, as a policy matter, always adhered to Presidential directives such as the Federal Radiation Guidance. While EPA does have the authority to establish regulatory standards for public health and environmental protection from all radioactive materials, this jurisdiction applies to environmental releases to areas outside the facilities regulated by NRC in the case of Atomic Energy Act materials.[298]

The existence of EPA's Federal Guidance role provides uniformity to worker protection from ionizing radiation, because several other agencies are also responsible for regulating occupational exposure to radiation. This complicated jurisdictional picture would otherwise result in a piecemeal approach to radiation safety. The Department of Energy, the Department of Defense, and the Department of Labor have regulations designed to indirectly limit certain exposures by regulating sources of exposure. The Department of Health and Human Services and the Department of Transportation indirectly regulate exposures.

NRC Regulations

NRC regulations do not explicitly address reproductive health, although it can be inferred from their structure and content that NRC

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[288] A Review of NRC Regulatory Processes and Functions, Advisory Committee on Reactor Safeguards, N.R.C. (1977).

[289] 42 U.S.C. § 2021(b)(3) (1984).

[290] 42 U.S.C. § 2021(c)(1) (1984).

[291] 42 U.S.C. § 2021(c)(2) (1984).

[292] 42 U.S.C. § 2021(c)(3)(4) (1984).

[293] 42 U.S.C. § 2021(d)(1)(2) (1984).

[294] Annual Report: 1983, U.S. Regulatory Commission (1983).

[295] Reorg. Plan No. 3 of 1970, 3 CFR § 1072 (1966-70 compilation), reprinted in 5 U.S.C. app. at 1132 (1982).

[296] Id.

[297] Radiation Protection Guidance for Federal Agencies, 25 Fed. Reg. 4402 (1970).

[298] Memorandum of Understanding, AEC-Licensed Facilities, 38 Fed. Reg. 24,936 (1973).

considered some aspects of reproductive health.[299] For example, the regulations deal with the sensitivity of youth, the various risks associated with cumulative dose, and the susceptibility of the gonads. **No regulations deal directly with the protection of the embryo/fetus, although a nonbinding regulatory guide advises women to minimize exposures while pregnant.** Thus, the NRC regulations must be carefully disassembled to determine their implicit application to the reproductive health of workers, and their adequacy for protecting reproductive health.

At the outset, it is instructive to understand the philosophical underpinnings of Federal regulation of occupational exposure. The regulation of radiation exposure encompasses two concepts: *the linear dose-response assumption* and the *"as-low-as-reasonably-achievable" (ALARA) assumption*.

Current analytic methods are not sensitive enough to define the pathological effects of chronic exposures to low levels of radiation. As stated in an NRC *Regulatory Guide,* "at the relatively low levels of occupational exposure in the United States, it is difficult to demonstrate correlations between exposure and effect."[300] In the absence of such evidence, the assumption is now made that there is no threshold dose below which radiation damage will not occur. Most authorities have therefore adopted the conservative hypothesis of a *linear relationship between dose and biological effect* even at very low doses. This means that each increment of radiation, however small, is currently assumed to inexorably result in an increment of health risk. This assumption determines Federal approach to the formulation of occupational radiation standards.

NRC espouses the "ALARA principle," which holds that despite the permissiveness of its standards, actual exposures should be kept "as low as reasonably achievable," and therefore at or below the level permitted by the standard.[301] This may be implemented in the design of facilities or through use of work practices that minimize unnecessary exposure.

These concepts are manifest in Part 20. The purpose of the regulation is to control the possession, use, and transfer of licensed material so that the total dose to a worker does not exceed the prescribed dose limit. The licensee is required to:

> . . . make every reasonable effort to maintain radiation exposures . . . as low as is reasonably achievable. The term "as low as is reasonably achievable" means as low as is reasonably achievable taking into account the state of technology, and the economics of improvements in relation to the benefits to the public health and safety, and other societal and socioeconomic considerations, and in relation to the utilization of atomic energy in the public interest.[302]

Three main sections of Part 20 are germane to reproductive health: 1) permissible doses, levels, and concentrations; 2) precautionary procedures; and 3) records, reports, and notification. (These are discussed in Appendix E.)

Applicability to Reproductive Risks

NRC has promulgated standards that implicitly account for many of the known reproductive sensitivities, and that represent what the Commission believes to be acceptable levels of risk. While both the International Commission on Radiation Protection (ICRP) and the National Council on Radiation Protection and Measurements (NCRP) have recommended lower occupational dose limits for fertile and pregnant women, criticism of these recommendations has prevented NRC from adopting differential exposure limits. Critics cite male reproductive susceptibility and carcinogenicity as supporting lower exposure limits for both sexes.

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[299] NRC regulations for protecting workers are set forth in 10 CFR pts. 19, 20 (1984). Part 19 establishes requirements for notices, instructions, and reports by licensees to employees who are exposed to radiation. Required procedures include instructions to the workforce concerning radiologic health protection, as well as reports to individual workers detailing their exposure. Part 20 defines permissible doses, levels, and concentrations to which employees can be exposed, and outlines precautionary procedures, including radiation surveys and personnel monitoring. Compliance with both parts 19 and 20 is a mandatory condition of all NRC licenses.

[300] Instruction Concerning Risk From Occupational Radiation Exposure, Regulatory Guide 8.29 and Value/Impact Statement, Office of Standard Development, Nuclear Regulatory Commission (1981).

[301] See discussion in Biological Effects of Ionizing Radiation, Report Nos. 1, 2, & 3, Advisory Committee on the Biological Effects of Ionizing Radiation, National Academy of Sciences (1972, 1977, 1981).

[302] 10 CFR pt. 20, § 20.1(c) (1975).

In 1984, the ICRP recommended that women of reproductive capacity should be employed only under conditions where the annual dose is unlikely to exceed 1.5 rems, delivered at an even rate. This would exclude any special permission to allow exposure up to 3 rems per quarter, as provided by NRC regulations previously discussed. It would also prevent high rates of exposure (i.e., exposure to the 3 rem quarterly limit in less than 3 months). The ICRP believed that these exposure conditions would keep the embryonic dose below 0.1 rem per month during the critical period of organogenesis. Once a pregnancy is diagnosed, ICRP also recommends that the women's exposure should be controlled so that the accumulated dose to the fetus during the remaining term does not exceed 0.5 rem, the upper limit for annual exposure of the general population.[303]

In 1971, the NCRP recommended that the dose to the fetus from occupational exposure of the mother not exceed a total of 0.5 rem over the period of gestation. This recommendation was similar to the then-current recommendation of the ICRP (1 rem). A statement accompanying the recommendation stated:

> The need to minimize exposure of the embryo and fetus is paramount. It becomes the controlling factor in the occupational exposure of fertile women. In effect, this implies that such women should be employed only in situations where the annual dose accumulation is unlikely to exceed 2 or 3 rems and is acquired at a more or less steady rate. In such cases, the probability of a dose to the fetus exceeding 0.5 rem before a pregnancy is recognized is negligible. Once a pregnancy is known, the actual approximate dose can be reviewed to see if work can be continued within the framework of the limit set above. . . . For conceptual purposes, the chosen dose limit essentially functions to treat the unborn child as a member of the public involuntarily brought into controlled areas. The NCRP recommends vigorous efforts to keep exposure of an embryo or fetus to the very lowest practicable level.[304]

In response to early ICRP and NCRP recommendations, the predecessor agency to NRC published proposed amendments to 10 CFR Parts 19 and 20 in 1975 that were designed to incorporate the "intent" of then-current ICRP and NCRP recommendations. It did not propose to amend the dose-limiting sections of the regulations, which would have resulted in differential standards for men and women. The proposed amendments would only require licensees to provide instructions to all workers that include information about the biological risks to embryos and fetuses exposed to radiation, and would require that women be advised of the need to keep exposures of the fetus to the very lowest practicable level during the entire gestation period.[305] These amendments were not adopted. While recognizing the greater radiosensitivity of the fetus, NRC did not believe a reduction of exposure limits for all workers was "practicable":

> Reduction of the dose limits for all radiation workers in order to avoid discrimination against women does not appear practicable. Such a reduction in the dose limits would cost the nuclear industry large sums of money in the application of design and engineering changes and, in some cases, the employment of additional workers in order to accomplish essential work within the reduced individual dose limits. The latter could even result in a net increase in total man rems of exposure.[306]

NRC also believed that actual exposure of pregnant women was currently within the NCRP recommendation, making adoption of the proposed changes unnecessary. It made this finding on the basis of mandatory licensee reports for 1973, which showed that 29,169 workers received measurable doses averaging 0.73 rem per year, and that 3,435 workers had exposures in excess of 2 rems, in industries believed to have the greatest worker exposures. NRC also assumed that many working women were not fertile, and that only a small portion of the fertile women being exposed would become pregnant. The Commission concluded that:

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[303] 14 Annals of the International Commission on Radiation Protection, No. 1 (1984).

[304] Review of the NCRP Radiation Dose Limit for Embryo and Fetus in Occupationally Exposed Women, Report No. 53, National Council on Radiation Protection and Measurements (1977).

[305] 40 Fed. Reg. 800 (1975).

[306] 40 Fed. Reg. 799 (1975).

> . . . the continued implementation of ALARA in its licensing and enforcement process . . . will result in further reduction in radiation doses, and may make specific adoption of the NCRP recommendation regarding additional limitation on exposure of fertile women of minor effect.[307]

The impact of the proposed amendment on women's privacy and employment opportunities also figured in NRC's informal decision to reject amending its regulations. The proposed amendment was instead made into an appendix to *Regulatory Guide Number 8.13*. The Guide instructs NRC licensees to instruct all workers about the biological risks to embryos and fetuses from radiation, and to advise women of the need to minimize exposures while pregnant. The Guide is nonbinding but considered persuasive.

A salient feature of NRC's exposure regulations is the failure to control the *rate of exposure*. While the regulations limit a worker's dose to a maximum of 3 rems per quarter, they do not prevent that exposure from being attained in minutes. It does not appear that the rate of exposure increases the risk for adult workers; 3 rems is believed to carry the same probability of genetic damage whether attained in minutes or in weeks. However, the failure to restrict the rate of exposure has two important implications for reproductive health. First, an acute exposure that coincides with the sensitive stages of embryonic or spermatogenic development can have a severe health effect even though the pregnant woman or prospective father may be well within the 3-rem-per-quarter dose limit. Second, NRC's failure to restrict the rate of exposure makes possible the use of *temporary workers* as a means of meeting exposure limits and circumventing the ALARA mandate.

Draft recommendations that would revise current Federal Radiation Protection Guidance would delete the 3 rems per quarter limit in favor of a 5 rems per year whole-body dose equivalent limit, believed to be sufficient to protect against the risk of lethal cancer and prompt genetic effects (those in the first two generations). It would also explicitly limit exposure of the fetus to 0.5 rem, and would recommend avoidance of variation above the uniform monthly exposure rate that would satisfy this limiting value. The draft recommendations state, as a matter of policy, that conformance to the limiting value for the unborn should be achieved without economic penalty or loss of job opportunity and security to workers. They also recommend that employees exposed to radiation be instructed as to the genetic and fetal health risks of exposure. These recommendations are expected to be transmitted to the President for approval in late 1985.

Temporary Workers[308]

A principal purpose for regulating occupational exposure to radiation should be the minimization of genetic risk to the population. This goal may be jeopardized if NRC licensees continue to be permitted to hire, quickly expose, and dismiss large numbers of temporary workers, also known as "sponges" or "jumpers."

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[307] 40 Fed. Reg. 800 (1975).

[308] Attention was first focused on the issue of temporary workers through investigations of a reprocessing and waste storage facility which was plagued by design defects and frequent breakdowns that resulted in high occupational exposures. During its 6-year history, the company employed about 170 full time workers, but in 1971 alone, 991 temporary workers were used. House Committee on Government Operations, West Valley and the Nuclear Waste Dilemma, H.R. Rep. No. 755, 95th Cong., 1st sess. (1977). Thirty percent of the occupational radiation exposure accrued to temporary workers, each of whom had less than one day's employment in the facility. Temporary workers would often receive a full quarterly dose in one day's work. Wages for less than 1 percent of the plant total went to temporary workers. R.W. Kates and B. Braine, The Locus of Benefits and Risks of West Valley Nuclear Wastes, Center of Technology, Environment, and Development, Clark University (1982). Some believe that this is unfair:

> Whether a worker receives his quarterly maximum of 3 rems in 3 months or in 3 minutes may make no biological difference. But if, as is generally assumed, every exposure carries some discrete risk of genetic damage or illness, then the full-time worker who earns 3 months' pay for 3 months' radiation benefits considerably more than the worker who accepts the same risk—knowingly or not—for half a day's pay. Gillette, Transient Nuclear Workers: A Special Case For Standards, Science 125 (Oct. 11, 1974).

This argument does not consider the fact that a nuclear worker's wages are based on the amount and type of labor as well as the amount of exposure, however. The typical temporary worker is paid substantially more, on an hourly basis, than other nuclear workers with similar skills, and this differential probably represents the market price of the difference in radioactive exposures.

The company discussed above represents an extreme case. But the employment of temporary workers as a means of meeting exposure standards is a permanent, prevalent, and growing nuclear industry practice. M.H. Melville, The Temporary Worker in the Nuclear Power Industry: An Equity Analysis, Center for Technology, Environment, and Development, Clark University (1981). See also 1984 Nuclear Power Safety Report, Public Citizen (1984).

> Jumpers are unskilled, short-term employees who expose themselves to quick doses of relatively high radiation for relatively high pay, often for only minutes of work. Chosen at the "body shop" for their small size, which enables them to crawl through the 18-inch-wide passageways of mammoth steel reactor pressure vessels, they may do no more than turn a bolt. But in a workplace giving off as many as 25 rems an hour of radiation, it must be done in seconds.[309]

The ALARA admonition does not make clear whether that concept requires *individual* exposures or *work force population* exposures to be as low as reasonably achievable when a choice between the two must be made. Industry's use of large numbers of temporary workers to perform tasks resulting in high exposures results in many workers being exposed to radiation (high population exposure), but to lower levels per capita than if a smaller number of permanent workers performed these tasks (high individual exposure). Although NRC regulations do not explicitly state which of the two types of exposures is preferable, high population exposure is implicitly preferred by the NRC regulations, since individual exposures are expressly limited while population exposures are not.

The use of large numbers of temporary nuclear workers may represent a public reproductive health problem, since brief but relatively high exposures to radiation may affect the workers' ability to parent healthy children if the reproductive safety threshold is relatively low. The Bulletin of Atomic Scientists has also expressed concern:

> The fact that many nuclear power plants are finding it necessary to solve the individual exposure problem of repair work in persistently high radiation areas by hiring temporary employees to spread out the dose has increased the overall cancer and genetic risks to the population, which is exactly what we should try to avoid.[310]

Concerned about temporary workers, NRC analyzed the mandatory annual reports filed by nuclear power companies. The reports showed several thousand employees had been hired and terminated more than once in 1977. The indicated periods of employment were less than 90 days in about half of the cases.[311] In an effort to monitor these employees, NRC focused on "transient workers," those employees hired and terminated by two or more employers in one quarter. NRC believes this class to be the most mobile and therefore the most vulnerable to overexposure.[312]

Between 1973 and 1977, the number of nuclear power workers exposed to measurable levels of radiation tripled to reach 71,904. Although the average level of exposure declined from 0.87 to 0.74 person rems per year, an eightfold increase occurred in the number of transient workers, from 157 to 1,311. The average exposure for these workers fell from 0.89 to 0.52 person rems per year.[313] Nevertheless, distributing small doses over an enlarged worker population may have effects on reproductive health in the Nation.

NRC's narrow definition of transient workers represents only a fraction of the temporary work force. When defined simply as the class of workers hired on any basis other than permanent, estimates of the size of the temporary work force are 18 times that of NRC's "transient workers." Under this definition, there were 23,520 temporaries in 1977, which represented 35 percent of the monitored work force. These workers received 47.5 percent of the radiation dose.[314]

The use of temporary workers presents a profound ethical question. Since a worker is part of the human gene pool, his dose is genetically significant for the entire population. Therefore, when a worker receives a radiation dose to the gonads, the worker and society are both harmed.

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[309] Nuclear Plants Hiring Stand-ins to Spare Aides Radiation Risks, N.Y. Times, July 16, 1979, at 1.

[310] K.Z. Morgan, Cancer and Low-Level Ionizing Radiation, The Bulletin of Atomic Scientists, 30 (September 1978).

[311] Occupational Radiation Exposure, Tenth Annual Report, 1977, Office of Management and Program Analysis, Nuclear Regulatory Commission (1978).

[312] M.H. Melville, The Temporary Worker in the Nuclear Power Industry: An Equity Analysis, Center for Technology, Environment, and Development, Clark University (1981). See also 1984 Nuclear Power Safety Report, Public Citizen (1984).

[313] M.H. Melville, The Temporary Worker in the Nuclear Power Industry: An Equity Analysis, Center for Technology, Environment, and Development, Clark University (1981).

[314] Id.

Given the linear dose-response assumption, genetic injuries are proportional to the dose received. A large dose to a limited number of workers can therefore have less effect on future generations and the entire society than small doses distributed across a larger work force. NRC regulations permit the widespread practice of hiring temporary workers; this practice defeats the purpose of radiation health protection. (A discussion of radiation regulation in Europe appears in a working paper available from OTA.)

Conclusions

NRC regulations[315] for protecting worker health do not explicitly address reproductive health, but manifest various reproductive health concerns in that they provide for special protection for the gonads and for various health risks to reproduction that arise from cumulative dose. No provisions deal with fertility, pregnancy, or protection of the embryo/fetus per se. However, NRC Regulatory Guide 8.13 provides information on risks. In developing its standards for worker protection, NRC employs a linear dose-response assumption. Furthermore, NRC requires its licensees to do more than merely comply with its standards, namely, to make every reasonable effort to maintain radiation exposures "as low as reasonably achievable" (the ALARA concept).

The exposure of regular employees (whole body; head and trunk; active blood-forming organs; lens of eye; or gonads) is limited to between 1.25 and 3 rems per calendar quarter, depending on the worker's accumulated lifetime dose from prior occupational exposures. Thus, employees are limited to 5 rems of radiation exposure per year. Workers under 18 years of age are more stringently protected, with the maximum dose to the minor's gonads set at 0.125 rem per calendar quarter.

In addition, NRC requires employers who have been licensed to handle radioactive materials to conduct various precautionary procedures, which also serve to safeguard reproductive health. These include periodic surveys of radiation hazards, use of personal monitoring equipment by workers, demarcation of restricted areas, maintenance of records of radiation surveys and personnel exposure, and furnishing of general instructions and individual exposure data to workers.

NRC standards are uniformly applied, irrespective of worker sex. NRC mandates no special protections for the fetus. The International Commission on Radiation Protection (ICRP) has recommended that women diagnosed as being pregnant be employed only where the annual dose is unlikely to exceed 1.5 rems, and not be permitted to receive the maximum 3 rems per quarter NRC regulations now provide for workers without records of prior occupational exposures. They further recommend that fetal protection should be "broadly comparable with that provided the general public" (i.e., 0.5 rem), and that substantial irregularities in the *rate* of exposure not occur. This would keep the fetal dose below 1 rem during the critical period of organogenesis.[316] The National Commission on Radiation Protection (NCRP) has recommended a protective limit of 0.5 rem for occupational exposure of women during the entire period of gestation. Controversy over these proposals exists.

NRC has not adopted these recommendations. According to its formal statement, it does not believe the recommendations are practicable, in that they would result in high costs for the nuclear industry and the employment of additional workers, which could even result in a net increase in total man rems of exposure. It has also provided further reasons: that actual exposure of pregnant women meets the NCRP recommendation; that the ALARA concept works to further reduce actual doses; and that the recommendations, if adopted, would lead to intrusions into the privacy of female workers and sex discrimination in violation of Federal law by their employers. NRC has, however, issued an appendix to one of its regulatory guides, which asks NRC licensees (employers) to instruct workers about risks to a fetus from radiation, and to advise women of the need to minimize exposure when pregnant.

Nor has NRC controlled the *rate of exposure* by regulatory action. This means that a pregnant woman, who may be well within the 3-rem-per-quarter dose limit for previously exposed workers, may be permissibly exposed to this quarterly limit in a matter of minutes. Such a focused exposure may coincide with the sensitive stages of embryonic development and have severe health effects.

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[315] 10 CFR pts. 19, 20 (1984).

[316] 14 Annals of the International Commission on Radiation Protection, No. 1 (1984).

NRC's silence on acute exposure with high dosage has also led to widespread use of temporary workers in industry as a means of meeting exposure limits while keeping individual doses relatively low over time. By 1977, temporary workers represented 35 percent of the work force in the nuclear power industry alone, with these workers receiving an estimated 47.5 percent of the total work force radiation dose.[317] Although quarterly dose limits are generally adhered to by the employers of temporary workers, temporary workers without occupational dose records are permitted to receive the higher doses of up to 3 rems per quarter, and, in practice, may receive this dose in a very short period of time (minutes), thereby endangering the embryo/fetus, as noted above. The distribution of small doses across an enlarged work force that tends to involve younger, temporary workers has resulted, and could have a greater impact on future generations than would a large dose to a smaller number of permanent workers.[318]

NRC authority, while preempting State law on matters involving health and safety regulations, does not preclude tort actions or workers' compensation by injured workers, under State law. Thus NRC licensees are subject to NRC standards and NRC license provisions, but may also be subject to private claims for compensatory and punitive damages by injured employees, their spouses, and their children, under circumstances that differ from State to State.

Finally, the factual basis for NRC regulatory actions on health issues has not been adequately tested in the courts. The Federal courts have repeatedly deferred to NRC expertise and discretion, and have failed to probe NRC technical findings and assumptions in affirming NRC regulatory decisions. Tort suits against the NRC have also failed to provide for accountability, since the courts have barred such suits on the grounds that NRC is exempt from Federal tort claims because its actions fall within the "discretionary function" exception of the Federal Tort Claims Act.[319]

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[317] M.H. Melville, The Temporary Worker in the Nuclear Power Industry: An Equity Analysis, Center for Technology, Environment, and Development, Clark University (1981). See also 1984 Nuclear Power Safety Report, Public Citizen (1984).

[318] K.Z. Morgan, Cancer and Low-Level Ionizing Radiation, The Bulletin of Atomic Scientists 30 (September 1978).

[319] 28 U.S.C. § 2680(a) (1982). See Dalehite v. United States, 346 U.S. 15 (1953).

Chapter 8

Sex Discrimination Issues

CONTENTS

Page
Introduction 235
Historical Perspective: The Common Law and Protective Legislation 235
The Equal Protection Clause and Discrimination 237
  Discrimination on the Basis of Pregnancy 240
Federal Statutes Relating to Sex and Pregnancy Discrimination in Employment 241
  Types of Discrimination 242
  Exceptions to the Prohibition Against Discrimination 242
The New Protectionism 244
  Hayes v. Shelby Memorial Hospital 245
  Zuniga v. Kleberg County Hospital 248
  Wright v. Olin Corp. 249
Case Study: American Cyanamid's Fetal Protection Policy 251
  American Cyanamid Co. Response 258
Conclusion 260
Technical Note 8.1: Litigation of Sex Discrimination Cases 261
Appendix 8A: Reproductive Health Protection Policies 263

List of Tables

Table No.
Page
8-1. Summary of Equal Protection Analysis 240
8-2. Summary of Types of Discrimination and Exceptions 244

List of Figures

Figure No.
Page
8-1. Summary of Discriminatory Treatment Litigation 243
8-2. Litigation of Fetal Protection Cases Under Hayes 249

Chapter 8

Sex Discrimination Issues[1]

INTRODUCTION

Some companies have implemented, or are considering, policies that exclude women of childbearing age from jobs involving exposure to suspected reproductive health hazards. Although it is impossible to determine how many companies have either written or unwritten exclusionary policies, at least 15 of the Fortune 500 as well as numerous hospitals are reported to exclude fertile and/or pregnant women from some jobs. Restricting the employment rights of women presents difficult ethical, legal, and policy questions. This chapter focuses on the legal aspects of sex discrimination and discusses the dilemma of balancing apparently competing policies of nondiscrimination and occupational health. (A discussion of the ethical aspects of sex discrimination appears in chapter 11.) The chapter begins with a historical view of exclusionary policies promulgated by State legislatures and implemented by employers. Special attention is paid to the ideological forces that have identified women as being hypersusceptible to occupational health hazards and once served as the basis for judicial approval of discriminatory policies. The chapter next addresses modern discrimination law and analyzes the law's ban on employment discrimination as it relates to exclusionary policies based on sex. The chapter concludes with a discussion of the relationship between Federal anti-discrimination law and the need to protect worker and fetal health.

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[1] References in the text to judicial and legislative bodies include both Federal and State institutions unless otherwise noted.

HISTORICAL PERSPECTIVE: THE COMMON LAW AND PROTECTIVE LEGISLATION

In 1869, Myra Bradwell applied for admission to the Illinois bar. Although she had passed the qualifying examination, she was denied admission by the State supreme court because she was a woman. Bradwell took her case to the Supreme Court of the United States, claiming she was unconstitutionally denied the privileges and immunities guaranteed to all citizens of the United States by the recently ratified 14th Amendment to the United States Constitution.[2] The Supreme Court rejected her claim. An opinion agreed to by three justices stated:

> [T]he civil law, as well as nature herself, has always recognized a wide difference in the respective spheres and destinies of man and woman. Man is, or should be, a woman's protector and defender. The natural and proper timidity and delicacy which belongs to the female sex evidently unfits it for many of the occupations of civil life. The constitution of family organization, which is founded in the divine ordinance, as well as in the nature of things, indicates the domestic sphere as that which properly belongs to the domain and functions of womanhood. The harmony, not to say identity, of interests and views which belong, or should belong, to the family institution is repugnant to the idea of a woman adopting a distinct and independent career from that of her husband. So firmly fixed was this sentiment in the founders of the common law that it became a maxim of that system of jurisprudence that a woman had no legal existence separate from her husband, who was regarded as her head and representative in the social state; and, notwithstanding some recent modifications of this civil status, many of the special rules of law flowing from and dependent upon this cardinal principle still exist in full force in most States. . . . The paramount destiny and mission of woman are to fulfill the noble and benign offices of wife and mother. This is the law of the Creator.[3]

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[2] Section 1 of the 14th amendment states:

> All persons born or naturalized in the United States, and subject to the jurisdiction thereof, are citizens of the United States and of the State wherein they reside. No State shall make or enforce any law which shall abridge the privileges or immunities of citizens of the United States; nor shall any State deprive any person of life, liberty, or property, without due process of law; nor deny to any person within its jurisdiction the equal protection of the laws.

[3] Bradwell v. Illinois, 83 U.S. (16 Wall.) 1130 (1873).

Bradwell was one of the first cases in which a woman went to court in an attempt to secure the freedom to choose an occupation. The opinion quoted here is representative of both judicial and societal attitudes of that era: a woman's role—first, foremost, and preferably exclusively—was that of wife and mother. Women were not supposed to work outside the home, and society saw the increasing numbers of working women as cause for civic concern and moral outrage.

Nevertheless, women began entering the nonagricultural labor force in large numbers in the 1880s. By the turn of the century, they constituted approximately 20 percent of the nonagricultural labor force.[4] The belief that women were inferior to men encouraged companies to use women workers only for the "women's work" that women had been doing in the home for centuries (e.g., sewing and weaving). The labor market was fundamentally segregated by sex; women were confined to the same few low-paying job categories as were reserved for children. Despite their marginal status as workers, women became a reserve force of inexpensive labor available to replace higher paid males in the nascent labor unions. They thus threatened men's jobs and wage levels, which may have helped motivate the suggestion that women stay home.[5] Yet, the fundamental sex segregation of the labor market was not affected by the occasional use of women to replace men simply because there were too few working women to replace men in any substantial numbers.

During the late 19th and early 20th centuries, labor unions often discriminated against women as much as employers did. Some union constitutions excluded women from membership, some set quotas on female membership, and others limited women to positions as apprentices or helpers. A few unions organized women into separate locals. Unions often negotiated contracts for women to be paid less than men and for women to be excluded from "men's jobs."[6] One labor historian has described the attitudes of unions as "a tacit understanding in the great brotherhood of man, that woman's place was in the home."[7] An American Federation of Labor pamphlet from this period stated this view quite directly:

> . . . as the woman is transferred from the home to the workshop . . . her refinement and elevating influence in the domestic circle [is] destroyed, and hence the social environment, and therefore the character of the child, the family, and ultimately that of the whole industrial community is thereby lowered.[8]

During this same period, working conditions resulting from the industrial revolution raised concerns about workplace healthfulness. The States began to enact laws, known as protective labor laws, regulating the working conditions for both men and women. Many of these statutes applied only to women, or required different working conditions for women. These laws limited the weights women could lift, the hours they could work, and the jobs they could perform; established a minimum wage for women; and generally attempted to protect the health and safety of women workers. Women's organizations, having failed to secure voting rights for women, launched a strategy of improving the status of women in other sectors of society and were prominent among those who lobbied in favor of protective legislation. Unfortunately, protective laws were often revealed to be ruses for "protecting" women from more lucrative jobs. For example, women were "protected" from lucrative night work in factories, but not night work as waitresses, and in California the maximum hours law for women was suspended during harvest season.

By 1908, 20 States had enacted laws setting maximum hours or prohibiting night work for women.[9] The constitutionality of these laws was upheld by State courts in four States and struck down in two States.

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[4] *Bread and Roses: Working Women's Consciousness Develops, 1905-1920*, 10 The Human Factor, Journal of the Graduate Sociology Student Union of Columbia University, No. 1, 33 (1970).

[5] B. Babcock, A. Freedman, E. Norton, and S. Ross, *Sex Discrimination and the Law: Causes and Remedies* 24 (1975).

[6] Falk, *Women and Unions: A Historical View*, Women's Rts. L. Rep. 54 (spring 1973).

[7] Wolfson, *Trade Union Activities of Women*, 143 Annals 123 (May 1929).

[8] Quoted in P. Foner, 3 History of the Labor Movement in the United States 224 (1947).

[9] Massachusetts, Rhode Island, Louisiana, Connecticut, Maine, New Hampshire, Maryland, Virginia, Pennsylvania, New York, Nebraska, Washington, Colorado, New Jersey, Oklahoma, North Dakota, South Dakota, Wisconsin, Oregon, and South Carolina.

Muller v. *Oregon*,[10] decided by the U.S. Supreme Court in 1908, was the the first case that involved a protective law affecting only women to reach the Supreme Court. The Court unanimously upheld Oregon's maximum hour rule for women, even though the Court had invalidated a New York protective law that established maximum hours for (generally male) bakers 3 years earlier.[11] The *Muller* decision stated that a woman "is properly placed in a class by herself, and legislation designed for her protection may be sustained, even when like legislation is not necessary for men and could not be sustained." The distinction between men and women was based in large part on scientific and pseudo-scientific data concerning the effects of overwork on "female functions," reproductive capacity, and infant mortality among the children of women workers. The *Muller* case was one of the first in which suspected reproductive impairment caused by working conditions was advanced as a justification to limit the employment of women. The Court justified the maximum hour rule by asserting that:

> . . . a "woman's physical structure and the performance of maternal functions place her at a disadvantage. . . . This is especially true when the burdens of motherhood are upon her. Even when they are not, by abundant testimony of the medical fraternity, continuance for a long time on her feet at work, repeating this from day to day, tends to [cause] injurious effects upon the body, and as healthy mothers are essential to vigorous offspring, the physical well-being of woman becomes an object of public interest and care in order to preserve the strength and vigor of the race.

After *Muller*, reform groups turned their attention to the establishment of a minimum wage for women and the issue was brought to the U.S. Supreme Court in 1923.[12] Supporters of the women's minimum wage statute submitted briefs filled with tables and charts demonstrating the impact of poverty and malnutrition on the health of women workers and their children. The Court, however, was unimpressed with arguments about the relationship between women's wages and the health of future generations and found the minimum wage law to be unconstitutional.[13]

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[10] 208 U.S. 412 (1908).

[11] Lochner v. New York, 198 U.S. 45 (1905).

[12] Adkins v. Children's Hospital of the District of Columbia, 261 U.S. 525 (1923).

[13] The courts continued to hold minimum wage laws, for both men and women, to be unconstitutional for almost 15 years, until the Supreme Court reversed itself. West Coast Hotel Co. v. Parrish, 300 U.S. 379 (1937).

THE EQUAL PROTECTION CLAUSE AND DISCRIMINATION

Basic constitutional principles control congressional and State legislative activity; congressional action that treats men and women differently for purposes of protecting fetal and adult health must meet constitutional standards. The equal protection clause of the 14th amendment is the primary constitutional limiting factor on legislating sex-biased classifications. The clause has no effect on the rights of the private sector to discriminate between men and women, though such discrimination might be a violation of the Federal sex discrimination statute (discussed later).

Historically, the courts have interpreted the equal protection clause as permitting almost any governmentally imposed restriction on the rights of women.[14] As in the case of protective labor legislation, women were considered to be special people whose morals, health, and childbearing capacity were in need of special protections and restrictions. Although the courts currently examine governmentally created sex-biased classifications much more closely than in the past, the courts are reluctant to equate the discriminatory potential of legislative classifications based on sex with those based on race or national origin. Consequently, women may continue to be subject to restrictions that would be unconstitutional if applied to a racial, religious, or ethnic group. This is the result of a judicially created theoretical framework that labels legislative classifications as either "suspect" (e.g., racial group) and therefore subject to a high level of judicial scrutiny, or "nonsuspect" (e.g., war veterans) and therefore subject to a low level of judicial scrutiny. Gender classifications were historically nonsuspect but now rank between these categories and are subject to "heightened scrutiny."

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[14] The 14th amendment is directly applicable only to the States and does not reach conduct by either the Federal Government or private entities. However, since the courts believe that equal protection concepts are an inherent part of due process, the substance of the equal protection clause has been made applicable to the Federal Government by incorporation into the due process requirement of the fifth amendment. Bolling v. Sharpe, 347 U.S. 497 (1954).

According to the courts, the equal protection clause does not require people or characteristics that are different to be treated by the law as though they were the same. For example, criminals need not be treated like law-abiding people, foreign nationals need not be treated like citizens, and children need not be treated like adults. But the courts do require that similar things be treated similarly. The judicially created *doctrine of reasonable classification* requires that legislative classifications such as these be reasonably related to accomplishing a constitutionally permissible purpose. A reasonable legislative classification should, so far as is possible, include all that is the same (lest it be underinclusive) and exclude all that is different (lest it be overinclusive). The extent to which a legislative classification is "reasonable" (and therefore acceptable to the courts) is determined by the classification's success in treating similarly those people who are similarly situated and excluding those who are not, given the legislative purpose of the classification.

For example, if a legislature wants to prevent birth defects caused by developmental hazards in the workplace (a constitutionally permissible purpose), it might decide to exclude from the workplace persons at risk. If the legislature excludes "all women," this classification might be *overinclusive* because it includes infertile women, who do not need protection from the risks of reproductive health hazards. However, excluding all women might also be *underinclusive* if men are subject to the same risk but have not been excluded. "All women" might also be considered overinclusive because it lumps together both women who are, or plan to be, pregnant with women who are practicing birth control or are abstaining and those who are no longer of reproductive age. Overinclusiveness and underinclusiveness are not necessarily mutually exclusive, nor is it always easy to determine the most appropriate classification that will achieve legislative goals.

After World War II, the reasonable classification test evolved into two alternative tests: the strict scrutiny test and the rational basis test. The choice of test is based on judicial labeling of a legislative classification as being either suspect or nonsuspect. Suspect classifications are subject to a stricter standard of review (strict scrutiny test) than are nonsuspect classifications (rational basis test).

A classification is suspect if it identifies for special treatment people who historically have been victimized by discriminatory treatment, especially if such people are easily identifiable by physical characteristics and are therefore easy targets of discrimination (e.g., race).[15] A classification labeled suspect is then subjected to a court's strict scrutiny and will be upheld only if the State shows: 1) that the legislative purpose is a "compelling State interest," meaning that the legislature's goal is of overwhelming public importance, and 2) the legislative purpose cannot be achieved with a less drastic classification than the one used. A less drastic classification would be less burdensome to the affected class, less underinclusive or overinclusive in defining the class, or would not use a suspect classification at all.[16]

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[15] Also, a law will be "suspect" if it infringes on an interest the courts deem to be "fundamental," such as the right to vote, the right to procreate, and the right to travel freely.

[16] In the example described previously, a strict scrutiny standard would prescribe a less drastic classification than "all women." A *less burdensome* law might require women to wear protective equipment or rotate job assignments rather than face expulsion from the workplace. If men are also at risk, a *less underinclusive* classification would include both men and women. A *less overinclusive* classification might be "all women between the ages of 16 and 45, except those who are certified infertile by a physician." A classification of "all women between the ages of 16 and 50 except those who are certified by a physician to be either (a) infertile, or (b) using an effective birth control method" would be even less overinclusive, but might be considered somewhat underinclusive because some women who use birth control become pregnant and are therefore subject to reproductive harm.

Legislative classifications that do not isolate a historically victimized group are labeled nonsuspect. For nonsuspect classifications, the courts require only that a "rational" relationship exist between the classification and a valid State interest. A "rational" relationship is one that is based on sufficient data to lead a court to conclude that the classification used is not arbitrary; it makes no difference that a more rational classification could have been chosen. Furthermore, the legislative purpose must merely be constitutionally permissible; a compelling State interest is not required.[17]

The difference between the strict scrutiny test (applied to suspect classifications) and the rational basis test (applied to nonsuspect classifications) is even greater than is immediately apparent. If a classification is nonsuspect, the person challenging the classification has the burden of proving to the court that the classification is arbitrary and has no rational basis. The courts ordinarily presume that the legislature is acting rationally and usually accept the legislature's version of the facts. Alternatively, if a classification is suspect, the legislature has the burden of proof on all issues, including whether the legislative purpose is a compelling State interest, whether the classification is necessary to achieve the legislative purpose, and whether less drastic alternatives to the classification are available.

Race is the quintessential suspect classification; members of minority racial groups have historically been discriminated against and have easily identifiable physical characteristics. Sex might have been labeled a suspect classification for the same reasons. However, the courts generally refused to analogize sex and race for purposes of choosing one of the two equal protection analytical frameworks, and, until recently, gender was considered a nonsuspect classification. The judiciary saw women primarily as mothers, wives, and homemakers, and as the morally pure members of the human species, and was as eager to "protect" women as were the legislatures. Until the late 1960s, the courts generally upheld sex-biased laws by applying the rational basis test.[18]

In the late 1960s, a number of cases brought to the lower courts challenged the notion that sex classifications were always reasonable. After the passage of Title VII of the Civil Rights Act of 1964, the courts began to examine more closely the States' justifications for differential treatment. In one case, a court refused to assume the existence of moral and social hazards in order to justify the exclusion of women from bars:

> Outdated images of bars as dens of coarseness and iniquity and of women as peculiarly delicate and impressionable creatures in need of protection from the rough and tumble of unwashed humanity will no longer justify separatism.[19]

In another bartending case, the California Supreme Court was the first State court to hold that sex was a suspect classification.[20] Decisions such as these in New York and California helped change judicial attitudes towards sex discrimination in other States and in the Federal courts.

The U.S. Supreme Court cautiously began breaking new ground in the application of equal protection analysis to sex discrimination in a 1971 case, *Reed* v. *Reed*.[21] *Reed* concerned a State law that gave mandatory preference to males over females as estate administrators, without regard to their individual qualifications. The Court unanimousy invalidated the law, holding that the preference for males was arbitrary and wholly unrelated to the objective of the statute (reducing the workload on probate courts). The Court applied neither the relatively deferential rational basis standard nor the sharper strict scrutiny standard, but rather a new approach somewhere between the two. This third approach recognized, for the first time, that a classification based on sex was subject to "scrutiny," but did not go so far as to require the legislature to have a "compelling State interest" or the classification to be the least drastic way of achieving the legislature's goals (see table 8-1).

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[17] If gender were a nonsuspect classification, then the classification in the previous footnote excluding "all women" may be constitutionally acceptable.

[18] For example, a State law that discriminated between male and female bar owners (by permitting the daughters of male bar owners to tend bar but not the daughters of female bar owners) was upheld by the Supreme Court in 1948. Applying the rational basis test, the Court held that the law was a permissible way to protect women from the "moral hazards" of dealing with drunken customers, even though the legislature chose to protect female bartenders by depriving them of their jobs rather than by penalizing antisocial customers. Goesaert v. Clearly, 335 U.S. 464 (1948). This decision was finally renounced by the Court in Craig v. Boren, 429 U.S. 190, 210 n.23 (1976). Less than a generation ago, a State supreme court upheld a statute excluding women from jury service with the following justification:

> The legislature has the right to exclude women so they may continue their service as mothers, wives, and homemakers, and also to protect them (in some areas, they are still on a pedestal) from the filth, obscenity, and noxious atmosphere that so often pervades a courtroom during a jury trial. State v. Hall, 187 So. 2d 861, 863 (Miss. 1966).

[19] Seidenberg v. McSorley's Old Ale House, 317 F. Supp. 593, 606 (S.D.N.Y. 1970).

[20] Sail'er Inn, Inc. v. Kirby, 5 Cal. 3d 1, 489 P.2d 529, 95 Cal. Rptr. 329 (1971).

[21] 404 U.S. 71 (1971).

In 1976, the Court clearly articulated a new standard for evaluating sex discrimination claims under the constitution. Classifications by gender are required to be "substantially related" to an "important Government objective," a stricter view than the rational basis test's "valid Government interest" but less stringent than the "compelling governmental interest" required under the strict scrutiny standard. Similarly, the classification itself was required to be "substantially related" to achievement of the legislative purpose; though this requires a more significant relationship than a mere "rational basis," the classification need not be the least drastic means of accomplishing the legislature's goals.[22] The "heightened scrutiny" test continues to be the standard against which most gender classifications are measured when challenged on constitutional grounds (as opposed to statutory grounds such as Title VII).

Discrimination on the Basis of Pregnancy

Pregnancy discrimination presents certain difficulties under historical equal protection analysis. The problem is an irreconcilable theoretical conflict between those who believe that the gender equality principle can be applied only where men and women are treated differently with respect to a shared characteristic (which pregnancy is not) and those who believe that discrimination on the basis of physical characteristics inextricably linked to one sex is a form of sex discrimination.[23] The courts have generally taken the former approach with the result that discrimination on the basis of pregnancy has *not* been deemed sex discrimination per se under constitutional analysis.

The Supreme Court was first urged to recognize pregnancy discrimination as sex discrimination in two 1974 cases.[24] Although the challenged law was invalidated in one case and upheld in the other, these cases made it clear that the Court believed that gender equality did not apply to cases where men and women are treated differently due to a difference in physical characteristics, rather than because of stereotypical notions as to the roles, abilities, and sensitivities of the sexes. These cases also demonstrated that the Court would continue to apply the rational basis test to pregnancy discrimination, rather than the middle ground test used in *Boren*.

In the *LaFleur* case, the Court held that school district rules requiring pregnant teachers to take unpaid maternity leave beginning 4 months before the expected childbirth were unconstitutionally burdensome on the "freedom of personal choice in matters of marriage and family life." Although the Court rested its decision on an interpretation of the due process clause rather than

Table 8-1.—Summary of Equal Protection Analysis

Type of classification	Test used	Legislative purpose must be:	Classification must be:
"Suspect" (example: race)	Strict scrutiny	Constitutionally permissible *and* of overwhelming public importance	Least drastic way to achieve purpose
Gender (since 1971)	Middle ground	Constitutionally permissible *and* important government objective	Substantially related to achieving purpose
"Nonsuspect" (including pregnancy and, before 1971, gender)	Rational basis	Constitutionally permissible	Rational way to achieve purpose

SOURCE: Office of Technology Assessment.

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[22] Craig v. Boren, 429 U.S. 190 (1976).

[23] B. Babcock, A. Freedman, E. Norton, and S. Ross, *Sex Discrimination and the Law: Causes and Remedies* (W. Williams Supp. 1978).

[24] Cleveland Board of Education v. LaFleur, 414 U.S. 632 (1974); Geduldig v. Aeillo, 417 U.S. 484 (1974).

the equal protection clause, the opinion employed an analysis similar to the rational basis test for nonsuspect classifications. The decision in *LaFleur* may be explained by the Court's increasing concern with the right to personal privacy in decisions relating to childbearing, as evidenced by its decision in a landmark abortion case the previous term.[25] The *LaFleur* policy assumed an irrebuttable presumption against a pregnant woman's fitness to teach.

In the *Geduldig* case, the Court upheld the validity of a State disability insurance system that excluded pregnancy from coverage, since the system did not exclude anyone from benefit eligibility because of gender but merely removed one physical condition—pregnancy—from the list of compensable disabilities. The Court used the rational basis test, refusing to equate pregnancy discrimination with sex discrimination in the absence of a showing that distinctions involving pregnancy are mere pretexts designed to effect sex discrimination. Three dissenting judges argued that the middle ground test should have been applied and the disability system invalidated:

> [B]y singling out for less favorable treatment a gender-linked disability peculiar to women, the State has created a double standard for disability compensation: a limitation is imposed upon the disabilities for which women workers may recover, while men receive full compensation for all disabilities suffered, including those that affect only or primarily their sex, such as prostatectomies, circumcision, hemophilia, and gout.

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[25] Roe v. Wade, 410 U.S. 113 (1973). In this case, the court held that a State criminal abortion statute that excempts from criminality only a *life-saving* procedure on behalf of the mother without regard to pregnancy stage and without recognition of the mother's personal privacy and other interests, is violative of the due process clause of the 14th amendment. The court attempted to balance the rights of a pregnant woman to preserve her health and privacy with the State's interest in protecting and preserving the health of both the pregnant woman and the "potentiality of human life." The court held that during the first trimester of pregnancy, the abortion decision and its effectuation must be left to the medical judgment of the pregnant woman's physician. For the stage running from the end of the first trimester until fetal viability, the State may, if it chooses, promote its interest in the health of the mother by regulating abortion procedures in ways that are reasonably related to maternal health. For the stage subsequent to viability, the State may, if it chooses, promote its interest in the potentiality of human life by regulating or even proscribing abortion except where it is necessary for the preservation of the life or health of the mother.

FEDERAL STATUTES RELATING TO SEX AND PREGNANCY DISCRIMINATION IN EMPLOYMENT

Title VII of the Civil Rights Act of 1964[26] prohibits sex discrimination by an employer of 15 or more persons engaged in any industry affecting commerce.[27] It is important to understand judicial interpretations of Title VII's requirements in order to understand the courts' treatment of exclusionary policies.

The principal language of the statute reads:

> It shall be an unlawful employment practice for an employer:
> 1. to fail or refuse to hire or to discharge any individual, or otherwise to discriminate against any individual with respect to his compensation, terms, conditions, or privileges of employment, because of such individual's race, color, religion, sex, or national origin; or
> 2. limit, segregate, or classify his employees or applicants for employment in any way which would deprive or tend to deprive any individual of employment opportunities or otherwise adversely affect his status as an employee, because of such individual's race, color, religion, sex, or national origin.

Because the statute did not define discrimination "because of sex," the Supreme Court was reluctant to expand its own narrow definition of sex discrimination so as to include pregnancy discrimination.[28] In a 1976 case that quoted *Gedul-*

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[26] 42 U.S.C. § 2000e (1982).

[27] Title VII does not apply to tax-exempt private membership clubs or to religious corporations, associations, educational institutions, or societies. In addition, the Federal Government is exempted from certain provisions of Title VII, though not from the prohibition against discrimination.

[28] Title VII's prohibition against sex discrimination was added as a floor amendment. As such, there is no committee report and very little legislative history to define the scope of that term. Some commentators believe the floor amendment was added in an attempt to defeat passage of the bill. M. A. Player, Federal Law of Employment Discrimination in a Nutshell (1976).

dig extensively, the Court held that a company did not violate Title VII by excluding pregnancy from its disability benefit plan. The Court again stated there that pregnancy discrimination was not the same as sex discrimination, unless a distinction based on pregnancy was in fact a "subterfuge" for sex discrimination.[29] The Court expanded the prohibition against pregnancy discrimination, however, in a later case.[30]

In 1978, Congress responded to the Court's refusal to categorize pregnancy discrimination as per se sex discrimination by amending Title VII to explicitly prohibit discrimination based on pregnancy. The amendment, known as the Pregnancy Discrimination Act, states:

> . . . [t]he terms "because of sex" or "on the basis of sex" include . . . because of or on the basis of pregnancy, childbirth or related medical conditions; and women affected by pregnancy, childbirth or related medical conditions shall be treated the same for all employment-related purposes . . . as other persons not so affected but similar in their ability or inability to work.

Types of Discrimination

Under Title VII, the courts use three analytical frameworks to analyze allegedly discriminatory policies.

The first framework applies in those situations in which the employer has engaged in "facial" discrimination. Facial discrimination occurs when an employer adopts a policy or practice of treating women differently than men because of their sex, such as excluding women from certain job categories. Such a practice is overtly and intentionally discriminatory; it is discriminatory on its face.

The second framework applies to those situations in which the employer adopts a policy or practice that on its face classifies workers on a neutral, nondiscriminatory basis, but which the plaintiff alleges to be a mere pretext for illegal discrimination. For example, an employer who is clever enough to avoid overt facial discrimination might impose neutral requirements which disproportionately affect women, solely as a ruse to effect intentional discrimination. Although the policy is neutral on its face, the employer's discriminatory motive makes this a pretext case.

The third framework is used when the plaintiff admits that the employer's policy is sex-neutral but seeks to demonstrate that the rule has a disproportionately adverse effect on women. The sex-neutral policy may be either a specific policy (e.g., height and weight minima) or a more general pattern of failing to hire women. Under this framework, neutral employment practices are judged by their impact and not by the good faith in which they were instituted. The absence of a discriminatory intent does not absolve an employer of Title VII liability. For example, a company might impose a height and weight requirement on its truck drivers. Since women are generally shorter and lighter than men, such a policy is facially neutral but has an adverse effect on women applicants. This policy would therefore be considered discriminatory.

Both facial discrimination and pretext cases are referred to as "discriminatory treatment" cases and require proof of the employer's intent to discriminate. Intent may be inferred from proof of the elements of a prima facie case (see figure 8-1). Cases involving a neutral rule with disproportionate adverse effects are known as "discriminatory impact" cases and do not require proof of a discriminatory motive (see table 8-2).

Exceptions to the Prohibition Against Discrimination

Title VII explicitly provides an exception to the prohibition against facial discrimination. The exception allows an employer to employ (or refuse to employ) an individual on the basis of sex, religion, or national origin where the individual's sex, religion, or national origin is a "bona fide occupational qualification [BFOQ] reasonably necessary to the normal operation of that particular business or enterprise." This BFOQ exception does not apply to facial discrimination on the basis of race or color, as these are never bona fide occupational qualifications under Title VII. The courts have created a similar exception for disparate impact cases so as to permit neutral rules that have a disparate impact when they are justified by

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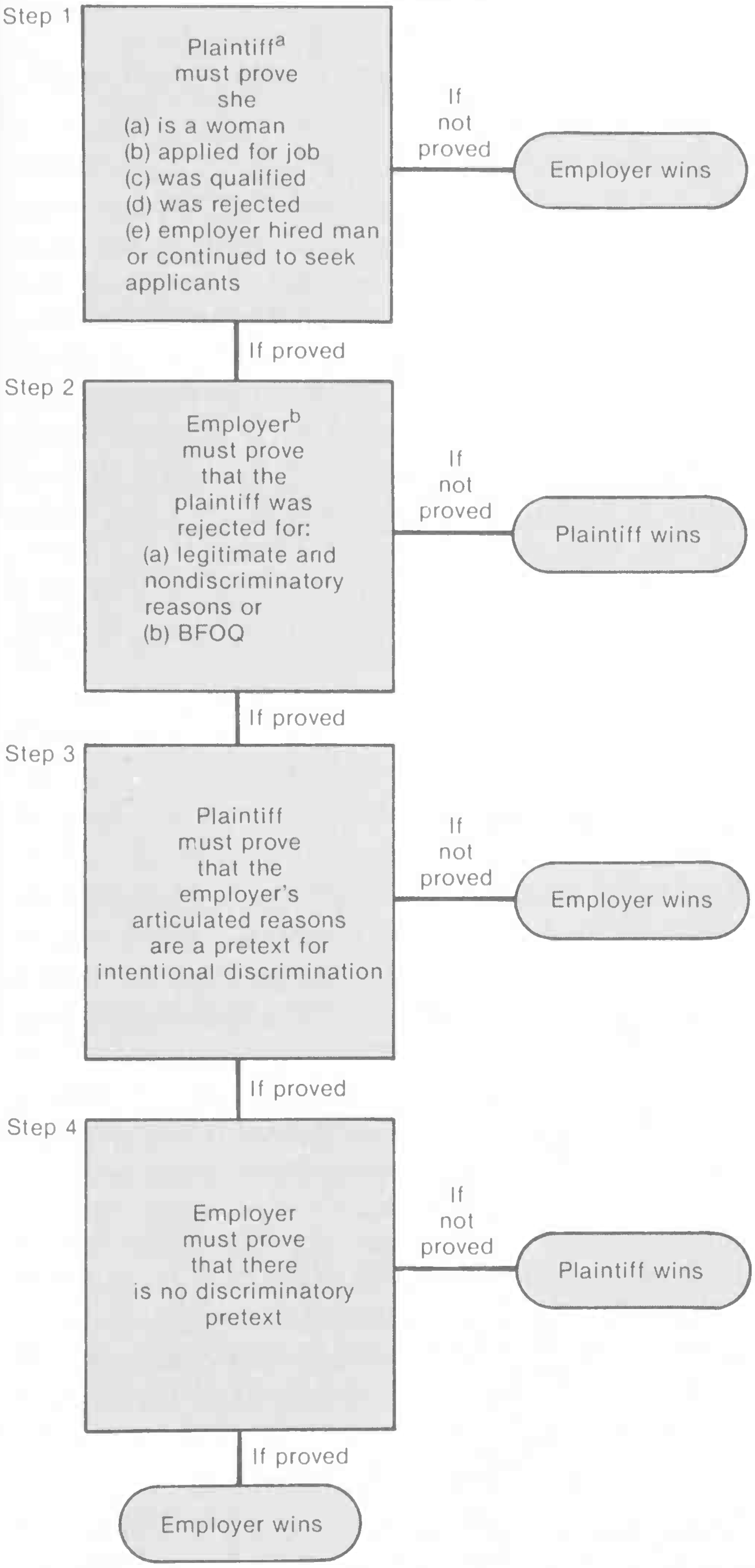
[29] General Electric Co. v. Gilbert, 429 U.S. 125 (1976).

[30] Nashville Gas Co. v. Satty, 434 U.S. 136 (1977).

Figure 8-1.—Summary of Discriminatory Treatment Litigation


Step 1
Plaintiff[a] must prove she
(a) is a woman
(b) applied for job
(c) was qualified
(d) was rejected
(e) employer hired man or continued to seek applicants
If not proved
Employer wins
If proved
Step 2
Employer[b] must prove that the plaintiff was rejected for:
(a) legitimate and nondiscriminatory reasons or
(b) BFOQ
If not proved
Plaintiff wins
If proved
Step 3
Plaintiff must prove that the employer's articulated reasons are a pretext for intentional discrimination
If not proved
Employer wins
If proved
Step 4
Employer must prove that there is no discriminatory pretext
If not proved
Plaintiff wins
If proved
Employer wins

[a] Plaintiff proves these elements when claiming she was refused a job on the basis of sex. Similar elements are proved in other types of facial discrimination cases.

[b] If the employer claims the plaintiff was rejected for legitimate and nondiscriminatory reasons, he is asserting that he did not discriminate. If employer claims BFOQ, he is claiming that he did discriminate but was justified in doing so.

Source: Office of Technology Assessment.

"business necessity." Unlike the BFOQ exception, the business necessity exception applies to policies that affect employees on the basis of race or color.

The BFOQ exception has been interpreted narrowly by the courts. Sex is a bona fide occupational qualification where it is genuinely essential for purposes of authenticity (e.g., requiring a female character to be portrayed by an actress) and successful job performance (e.g., requiring wet nurses to be female and sperm donors to be male), including safe operation of the business where safety is essential to the business (e.g., requiring a violent male prison population to be supervised by male guards).

Generally, however, the principle of nondiscrimination requires that individuals be considered on the basis of individual capabilities and not on the basis of any characteristics generally attributed to the group. The BFOQ exception does not permit sex discrimination because of customer preferences (e.g., an airline hiring policy reflecting customer preferences for male pilots and female stewardesses), assumptions about the comparative employment characteristics of women in general (e.g., the assumption that the turnover rate among women is higher than among men), or because of stereotypical characterizations of the sexes (e.g., that women are less capable of aggressive salesmanship). If a job requires, for example, regular lifting of heavy weights, an employer cannot refuse to consider women job applicants even though *most* men can perform this task more safely and efficiently than *most* women. Unless the employer can prove that *all or substantially all* women are unable to safely and efficiently perform the duties of the position, the employer is required to test each job applicant, male and female, to determine whether that particular individual is capable of performing the job.[31] Generally, the increased economic cost of testing women (or providing restroom facilities) may not be used to justify discrimination.

The exception in discriminatory impact cases is known as the business necessity exception. The

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[31] Rosenfeld v. Southern Pacific Co., 444 F.2d 1219 (9th Cir. 1971); Weeks v. Southern Bell Telephone & Telegraph Co., 408 F.2d 228 (5th Cir. 1969).

Table 8-2.—Summary of Types of Discrimination and Exceptions

Type of discrimination claimed:	Must plaintiff prove discriminatory intent?	Exception permitting discrimination:	Exception applies to:
Discriminatory treatment (facial and pretext discrimination)	Yes	Bona fide occupational qualification	Sex, religion, national origin (but not race or color)
Discriminatory impact (disparate impact)	No	Business necessity	Sex, religion, national origin, race, and color

SOURCE: Office of Technology Assessment.

exception is broader in definition than the BFOQ exception because it focuses on the general business enterprise and job-relatedness rather than the narrower concept of job qualifications.

For a policy to be a "business necessity," the business purpose must meet three tests. First, it must be sufficiently compelling to override any discriminatory impact. Second, the challenged policy must effectively carry out the business purpose. And finally, there must be no acceptable alternative policies that would be less burdensome to the protected class. Using this standard, the courts have decided that the following employment criteria are permissible in at least some circumstances and for some jobs, even though they have a disproportionately adverse impact on some groups: educational minima, seniority systems, strength and agility tests, height and weight minima, lack of criminal record, and previous experience.

THE NEW PROTECTIONISM

Thus far, this chapter has described the restraints historically placed on women's occupational choice by State legislatures and employers concerned with the possible adverse effects of work on women's health, offspring, mortality, and morality. Protective labor legislation was consistently upheld against constitutional challenges until the late 1960s and early 1970s, when the courts refused to continue to accept stereotypical characterizations of the "weaker sex" as adequate justification for overtly discriminatory policies. The enactment of Title VII provided impetus for this change in judicial attitudes towards State-legislated sex discrimination, as well as being the first Federal statute prohibiting discrimination by employers.

During the past 16 years, the courts have interpreted and reinterpreted the prohibitions of Title VII with increasing breadth, especially following passage of the Pregnancy Discrimination Act in 1978. The courts now consider disparate impact, pregnancy discrimination, and sexual harassment to be aspects of sex discrimination. Several States have passed amendments to their State constitutions affirming the right of women to receive equal treatment at the hands of employers. Furthermore, numerous employers have voluntarily or by court order established affirmative action programs to increase the number of female employees at all levels. Although vestiges of past discrimination remain (women continue to earn 60 to 65 percent as much as men do),[32] many barriers to occupational choice have been broken.

Given both the history of sex discrimination in the United States and the remarkable progress that has been made in the past decade, many people find it troubling that sex is once again the basis for exclusion from some workplaces due to the presence of known or suspected reproductive health hazards.

Company policies excluding either fertile or pregnant women from certain jobs are becoming increasingly common. The spectrum of employers instituting such policies ranges from large chemical and automobile manufacturers to small community hospitals.

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[32] Shack-Marquez, *Earnings Differences Between Men and Women: An Introductory Note*, U.S. Department of Labor, Bureau of Labor Statistics, Monthly Lab. Rev. (June 1984).

There is tremendous diversity in company exclusionary policies. Some of these policies have a basis in epidemiological and toxicological research findings with respect to particular substances, while others are more speculative about potential reproductive health hazards. Some policies are written and documented, while others are unwritten, making them more flexible but also more ambiguous. In large manufacturing companies, policies are generally announced to employees and their unions prior to implementation, while smaller organizations appear to formulate and apply policies as a perceived problem arises. Some policies recognize that a fetal hazard may be mediated through the male or female worker, while others by their terms apply only to women.

In some cases, these policies have faced court challenges on grounds of sex discrimination. While many of these cases are apparently settled out of court, some cases have been adjudicated. Three of these cases,[33] as noted in the following discussion, have reached the Federal courts of appeals in the Fourth, Fifth, and Eleventh Circuits. All three courts of appeals have held that the exclusion of fertile or pregnant women constitutes illegal sex discrimination under some circumstances, although these courts have approached the issue of exclusionary policies somewhat differently. One issue of disagreement is whether an employment policy barring pregnant or fertile women from certain job categories should be evaluated as sex-biased on its face (facial discrimination) because its terms apply only to women, or sex-neutral (disparate impact) because the policy's effect is similar on both sexes by providing equal health protection (though it may in fact be discriminatory by putting a disproportionate burden on women). The reason for this issue is that the choice determines whether BFOQ or business necessity is the relevant defense. Another point of contention has been whether an employer's concern about either fetal health or possible tort liability constitutes the business necessity defense. One circuit court treated fetuses like business visitors for purposes of determining the employer's responsibility for fetal safety.

Although the three courts used different approaches, the following general principles can be extracted from these cases:

- A fetal protection policy (FPP) that applies only to women is presumptively discriminatory. That is, the mere existence of an FPP will create Title VII liability for the employer in the absence of strongly supportive scientific evidence.
- To overcome the presumption of discrimination, the employer must be able to prove that the body of scientific evidence supports legal findings that: 1) exposure at the level encountered in the workplace involves a significant risk of harm to the unborn children of women workers, 2) exposure at the level encountered in the workplace does not involve a similar risk of harm to the unborn children of male employees, and 3) the FPP is effective in significantly reducing the risk. An employer's subjective but scientifically unsupportable *belief* in the necessity of the policy is insufficient to defend it.
- If the employer proves both points (embryo/fetal risk through maternal exposure and lack of embryo/fetal risk through paternal exposure), the plaintiff may nevertheless prevail by proving that an acceptable alternative policy would promote embryo/fetal health at least as well with a less adverse impact on one sex or by showing that the FPP is a pretext for discrimination.

Following is a description of the three cases decided by the Federal courts of appeals for the Fourth, Fifth, and Eleventh Circuits. The most recent decision, *Hayes* v. *Shelby Memorial Hospital*, is also the most analytically sound and the most likely to be followed by those jurisdictions that have not yet examined the issue of fetal protection as sex discrimination. The *Hayes* case is therefore discussed first and in greater detail than the other cases.

Hayes v. Shelby Memorial Hospital[34]

In August 1980, an Alabama hospital hired a female X-ray technician to work the night shift in the hospital's radiology department. Two months later, the technician was fired after she informed her supervisor that she was pregnant. Following her dismissal, the technician filed a sex discrimination suit against the hospital in Federal court. The hospital defended on the grounds of "bona fide occupational qualification" and "business necessity." The trial court concluded that the hospital violated Title VII and awarded the technician damages. The hospital appealed the decision to the U.S. Court of Appeals for the Eleventh Circuit. The appellate court examined the case under both facial discrimination and disparate impact theories, and concluded that the hospital had indeed violated the Federal statute governing sex discrimination.[35]

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[33] Hayes v. Shelby Memorial Hospital, 726 F.2d 2095 (llth Cir. 1984); Wright v. Olin Corp., 697 F.2d 1172 (4th Cir. 1982); Zuniga v. Kleberg County Hospital, 692 F.2d 986 (5th Cir. 1982).

[34] 726 F.2d 2095 (11th Cir. 1984).

The *Hayes* decision approaches the issue of fetal protection policies in a manner more consistent with traditional Title VII analysis than the other cases that have been decided by Federal appellate courts. The court of appeals began by establishing a presumption that if an employment policy by its terms only applies to women or pregnant women, then the policy is facially discriminatory. That presumption may be rebutted if the employer can show that, although its policy applies only to women, the policy is both necessary and neutral in the sense that it effectively and equally protects all employees. Thus, in a fetal protection case, the employer must meet the requirements of a two-pronged test. The employer must show: 1) that there is an unreasonable risk of harm from exposure to toxic hazards in the workplace to the fetuses of women employees during pregnancy, and 2) that the hazard applies to pregnant women, but not to men.[36] The court did not consider application of a fetal protection policy to nonpregnant women. Under the court's analysis, the burden of proving a substantial risk of harm to the fetus is a threshold requirement. To meet this burden, the employer must "produce objective evidence of an essentially scientific nature supported by the opinion evidence of qualified experts in the relevant scientific fields." This burden may not be carried by merely proving that the employer subjectively and in good faith believed a substantial embryo/fetal risk to exist. The employer need not show that a consensus exists within the qualified scientific community. Rather, the employer carries its burden by showing that "the body of opinion believing that significant risk exists is so considerable that an informed employer could not responsibly fail to act on the assumption that this opinion might be the accurate one."[37]

If the employer proves that there is a significant risk of harm to a developing fetus, it must then also prove that there is no similar risk for the offspring of male employees. Again, scientific evidence is necessary. The court noted that a "certain amount of subtle bias" has focused scientific research on hazardousness to the reproductive systems of women more so than on the hazards to male reproduction. Although the issue was not raised in the case, and is therefore still open to resolution, the court suggested that in those instances where scientific evidence points to a hazard to women, but no scientific evidence exists regarding men, an employer may be allowed to adopt a policy aimed solely at women. Presumably, however, employers would be required to adopt nondiscriminatory alternatives if available. and the failure to do so would be evidence of a discriminatory pretext.

If an employer fails to prove that the ultimate effect of a sex-based FPP is in fact sex-neutral in that it provides equivalent health protection to both sexes (due to both substantial risk to women and the absence of substantial risk to men), then the employer's only remaining defense is BFOQ. Utilizing the traditional analysis, the court stated that the BFOQ defense is available only when the employer can show that pregnant women are "unable to perform the duties that constitute the essence of the job."[38] Under this analysis, potential for embryo/fetal harm is irrelevant to the BFOQ issue unless the toxic exposure adversely affects a woman's job performance (e.g., by making her too afraid to perform her job). Thus, there is in effect no BFOQ defense unless the employer shows a direct relationship between the fetal protection policy and the actual ability of a pregnant woman to perform her job. Critics of this analysis assert that a sex-based policy cannot be converted into a sex-neutral one based on the policy's ultimate effect of protecting the offspring of both sexes.

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[35] *Id.* at 2108.

[36] *Id.* at 2101.

[37] *Id.*, quoting Wright v. Olin Corp.

[38] *Id.* at 2102.

Applying this framework to the facts of the *Hayes* case, the court found that a presumption of facial discrimination existed because only pregnant X-ray technicians were subject to removal from jobs requiring radiation exposure.

The court then turned to the issue of whether the hospital rebutted the presumption of discrimination. The court first looked at whether the hospital proved that radiation from X-rays posed a significant risk of harm to the technician's fetus. The expert witnesses generally agreed that the standards set by the National Council on Radiation Protection and Measurements (NCRP) were authoritative, conservative, and provided a wide margin for safety. The NCRP proposes 0.5 rem as the maximum radiation dose to which a fetus should be exposed during the 9 months of gestation. The technician's radiation badges, which monitored the amount of radiation to which she was exposed, indicated that the technician's total radiation exposure during pregnancy would be below the 0.5 rem limit. The evidence at trial led the court to conclude that, "although *any* amount of radiation *can* have a detrimental effect on humans, it is extremely unlikely in most cases that radiation below certain doses *will* have a detrimental effect"[39] (emphasis in original). The court concluded that the hospital had failed to prove that the technician's level of exposure posed an unreasonable risk of harm to her fetus.[40]

The court held that the hospital's failure to prove the necessity of its policy was sufficient to make the policy legally discriminatory. Having reached this conclusion, the court did not need to decide the factual issue of whether X-ray radiation affects the offspring of employees only through pregnant women, or whether similar effects can occur from male exposure.[41]

Although the court's decision rested on a facial discrimination analysis, the court also analyzed the case using disparate impact analysis to show that, even if a fetal protection policy is facially sex-neutral, the policy might still constitute illegal discrimination.

The court began its disparate impact analysis by assuming, for the sake of analysis, that the application of a fetal protection policy solely to pregnant or fertile women was scientifically justified under the two-pronged test requiring necessity (exposure of pregnant or fertile women would result in an unreasonable risk of harm to fetuses) and neutrality (exposure of fertile men would *not* result in an unreasonable risk of harm to fetuses). Such a policy would be facially sex-neutral but would nevertheless have a disproportionate impact on women as a class since only women are affected by the policy. Therefore, "even if the employer rebuts the prima facie case of facial discrimination, the employee has an automatic prima facie case of disparate impact."[42]

The *Hayes* court stated that the employer's business necessity defense, like the employee's prima facie disparate impact case, also applies "automatically" in fetal protection cases. This is because the employer, in rebutting the presumption of facial discrimination that necessarily precedes disparate impact analysis in a fetal protection case, has already proven that its policy is scientifically justified.

The court, by accepting scientific evidence of a *fetal* hazard as a basis for the business necessity defense, extended the defense beyond the traditional definition of business necessity. The traditional definition generally limits the application of the business necessity defense to situations in which adverse job performance makes an employment policy necessary, despite its disparate impact on a protected class. The court did, however, limit its extension of the business necessity defense by carefully limiting the defense to an employer's genuine desire to promote the health of its employees' offspring. Designating fetal protection as a "legitimate area of employer concern to which the business necessity defense extends,"[43] the court distinguished between the avoidance of potential tort liability (discussed in chapter 10) and concern for fetal health. The purpose of this distinction was to make clear that extension of the business necessity defense was "based on a higher public policy than simply protecting employers from lawsuits."[44] Although the hospital claimed that concern about the potential economic consequences of tort liability constituted a business necessity, the court rejected this argument for fear that such an extension of the defense would shift the focus of the defense from a concern for the safety of hospital patients to a concern for hospital finances.[45]

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[39] Id. at 2104.

[40] The court noted that, even if the hospital had proved that the technician's exposure was excessive, the fetal protection policy would probably have been ineffective because the greatest danger of fetal damage from radiation occurs during the earliest days of pregnancy. In such a case, the employee could reasonably assert that the policy was a pretext for discrimination.

[41] The court did note, however, the existence of studies suggesting that radiation-induced mutations can pass to offspring via the father's sperm.

[42] *Id.* at 2106. When the court says that the employee's case of disparate impact and the employer's defense of business necessity apply "automatically," this means that no additional evidence needs to be introduced at trial on these points, and the trial judge may proceed to the next issue, whether there were acceptable alternative policies.

The *Hayes* decision indicated that the employee may rebut the employer's business necessity defense with proof that there are "acceptable alternative policies that would better accomplish the purposes of promoting fetal health, or that would accomplish the purpose with a less adverse impact on one sex."[46] The burden of proving the existence of acceptable alternative policies rests on the employee. Such policies might include temporary reassignment, temporary change in job description, job rotation, engineering controls, substitution of materials, and use of personal protective equipment. If there is more than one possible alternative policy, the employer must adopt the most effective policy possible with the least disparate impact possible to avoid Title VII liability. Furthermore, evidence of either failure to consider nondiscriminatory alternative approaches to fetal protection or lack of concern for nonreproductive occupational health protection could be used to show pretextual discrimination.

Unlike most sex discrimination cases (which proceed under either facial discrimination, pretext discrimination, or disparate impact theory), cases involving fetal protection policies that apply only to women would proceed under both theories in a sequential manner under the *Hayes* approach. Since the employee's prima facie case of disparate treatment and the employer's business necessity defense are automatic, the employee's failure to prove facial discrimination would lead directly to the issue of alternative policies, as demonstrated in figure 8-2.

Zuniga v. Kleberg County Hospital[47]

Zuniga was another case concerning a hospital's firing of a pregnant X-ray technician. Unlike *Hayes*, the events in *Zuniga* all occurred prior to the effective date of the Pregnancy Discrimination Act.[48] Thus, under applicable Supreme Court precedent, a pregnancy-based distinction could not be characterized as facial discrimination. Nevertheless, the court found the policy to be discriminatory because of its impact on women, and held that no defense was made because the hospital failed to employ an "available, alternative, less discriminatory means of achieving its business purpose."[49] In this case, the less discriminatory policy was to grant the plaintiff a requested leave of absence in accordance with the hospital's own established policies. Although the court did not explicitly state whether the burden of proving the existence of a less discriminatory alternative falls on the plaintiff or the employer, the plaintiff in this case assumed the burden and won the case.

The *Zuniga* court did not decide whether concern over embryo/fetal health and fear of tort liability ever justifies termination on the basis of business necessity. The opinion suggests that the health of the embryo/fetus is more the concern of the mother than of the employer, and cites conflicting authority as to whether the economic consequences of a tort suit might constitute a business necessity for a fetal protection policy.[50] This

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[43] *Id.* at 2106 n.14.

[44] *Id.*

[45] *Id.* at 2106 n.15.

[46] *Id.* at 2107.

[47] 692 F.2d 986 (5th Cir. 1982).

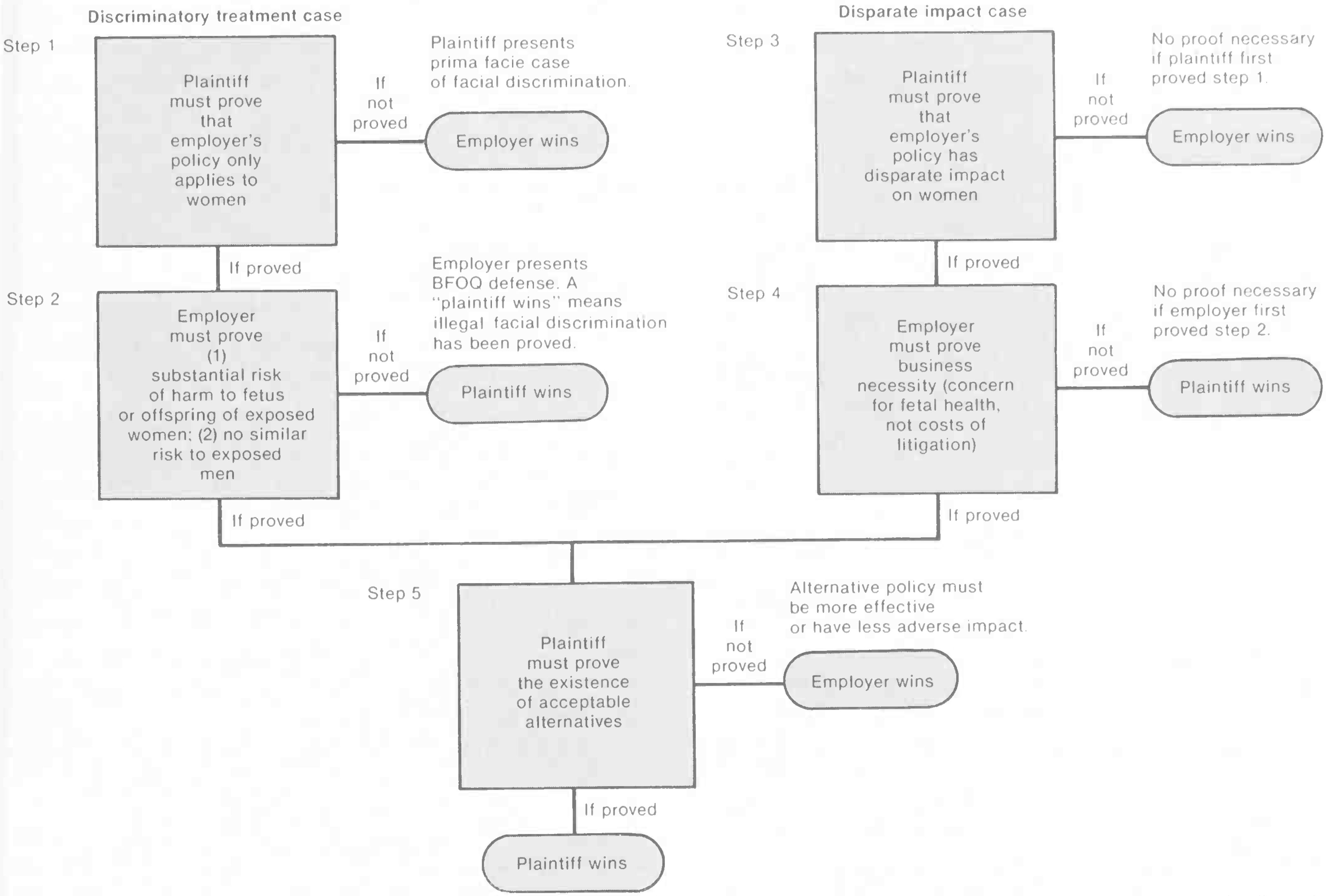
[48] 42 U.S.C. § 2000e(k)(1982). The Pregnancy Discrimination Act does not apply retroactively.

[49] 682 F.2d at 992.

[50] Pretextual discrimination is said to exist when a facially neutral rule disguises an employer's "hidden agenda" to intentionally discriminate. Because pretext cases are essentially cases of discriminatory treatment rather than disparate impact, they are judicially treated in accordance with their true nature (intentional discrimination) rather than their guise (disparate impact). For this reason, pretextual discrimination is only excusable when membership in a certain class is a BFOQ, and not merely when class membership is a business necessity. The distinction is important, as BFOQ is quite narrowly defined by the courts as limited to occupational qualifications genuinely necessary for successful job performance. Because BFOQs must be strictly performance-related, employer concerns about fetal health or tort litigation costs would never constitute BFOQs, though they might qualify as business necessities.

[55] *Id.* at 992 n.10.

Figure 8-2.—Litigation of Fetal Protection Cases Under Hayes


Discriminatory treatment case
Step 1
Plaintiff must prove that employer's policy only applies to women
If not proved
Plaintiff presents prima facie case of facial discrimination.
Employer wins
If proved
Step 2
Employer must prove (1) substantial risk of harm to fetus or offspring of exposed women; (2) no similar risk to exposed men
If not proved
Employer presents BFOQ defense. A "plaintiff wins" means illegal facial discrimination has been proved.
Plaintiff wins
If proved
Disparate impact case
Step 3
Plaintiff must prove that employer's policy has disparate impact on women
If not proved
No proof necessary if plaintiff first proved step 1.
Employer wins
If proved
Step 4
Employer must prove business necessity (concern for fetal health, not costs of litigation)
If not proved
No proof necessary if employer first proved step 2.
Plaintiff wins
If proved
Step 5
Plaintiff must prove the existence of acceptable alternatives
If not proved
Alternative policy must be more effective or have less adverse impact.
Employer wins
If proved
Plaintiff wins

Note: A facial discrimination case begins at step 1 and proceeds through step 2. If the plaintiff fails to win by the end of step 2, the case becomes one for disparate impact. However, no evidence need be introduced at steps 3 and 4 because these steps are "automatically" completed under the evidence presented at steps 1 and 2, respectively. A disparate treatment case begins at step 3 and proceeds through the remaining steps, with evidence introduced at each step.

Source: Office of Technology Assessment.

is distinct from *Hayes*, in which the court rejected the notion that economic consequences might constitute business necessity in fetal protection cases.

Wright v. Olin Corp.[51]

The first fetal protection case to reach a Federal court of appeals was *Wright* v. *Olin* in 1982, a class action suit charging the chemical company with race and sex discrimination. One of the issues was the legality of Olin's "fetal vulnerability" program, adopted in early 1978 after some 4 years of planning.

As required under Occupational Safety and Health Administration (OSHA) regulations, Olin orally warns its male employees about the dangers of lead, but the warnings are much less formal than the written warnings to women. In addition, while no restrictions are placed on male employees, Olin's fetal protection policy (FPP) excludes all unsterilized females between the ages of 5 and 63 from certain jobs.[52] Since only 1 out of every 5,000 women between the ages of 45 and 49 gives birth each year, and births to women between the ages of 50 and 63 are virtually nonexistent,[53] Olin's fetal protection policy is unnecessarily restrictive even if a fetal hazard exists.

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[51] 697 F.2d 1172 (4th Cir. 1982).

[52] "Restricted jobs" are those that Olin believes "may require contact with and exposure to known or suspected abortifacient or teratogenic agents." All women between the ages of 5 and 63 are excluded from such jobs, unless consultation with Olin's staff physicians confirms that a woman is sterile and will sustain no adverse health effects from exposure. "Controlled jobs" may require very limited contact with hazardous chemicals. Originally, all pregnant women were prohibited from working in such jobs. Several weeks later, Olin revised its policy to allow for a case-by-case review. Olin encourages women in controlled jobs to bid for other jobs if they intend to become pregnant. "Unrestricted jobs" are those that do not, according to Olin, present a hazard to the pregnant female or the fetus, and are open to all women. *Id.* at 1182.

The trial court ruled in favor of Olin, saying the FPP was based on sound scientific evidence, and that it was instituted and maintained with no intent to discriminate on the basis of sex. The plaintiffs appealed the decision to the U.S. Court of Appeals for the Fourth Circuit. The appellate court set aside the portion of the judgment applying to the FPP and remanded the case to the trial court for further factual development under legal principles, discussed below, that the appellate court held were not properly applied.[54] (After the case was remanded, plaintiff Wright moved for a voluntary dismissal on the grounds that her own claim was moot and that she was no longer a proper class representative. The trial judge refused to dismiss the case and a trial was held in which only Olin participated. The judge rendered another judgment favorable to Olin which has been vacated on constitutional grounds.)[55]

The appellate court decision conceded that the Olin FPP was "as a matter of law a *prima facie* Title VII violation," which is essentially the definition of facial discrimination.[56] Nevertheless, the court explicitly rejected facial discrimination/BFOQ analysis because the narrowness of the job performance-oriented BFOQ defense would almost always prevent the employer from asserting that an FPP is justified.[57] The court concluded that disparate impact/business necessity theory was more suited for application to FPP cases than the discriminatory treatment analysis applied by the trial court.

The appellate court attempted to divine probable congressional intent in its adaptation of the business necessity defense to FPPs.[58] The court began by asking whether fetal protection could under any circumstances be properly considered a business necessity. While the safety of women workers themselves might be thought to be the most obvious subject of legally justifiable employment restrictions, the opposite is the case. As the court noted, "it is the purpose of Title VII to allow the individual woman to make [the] choice for herself."[59] The same overriding consideration does not, however, apply to the safety of others. As the court stated, the safety of customers has been recognized as being sufficiently necessary to override Title VII considerations.[60]

The court compared the safety of embryo/fetuses to the safety of business customers and held that an employer may, as a matter of business necessity, impose otherwise impermissible restrictions on female employment that are "reasonably required to protect the health" of embryo/fetuses.[61] The court stated that the business necessity was based on a "general societal interest" in having business enterprises operated in ways that preserve the health of workers and consumers, rather than on the avoidance of potential tort liability.[62]

The other principles that the court deemed to be controlling in FPP cases were substantially repeated in *Hayes*. According to *Olin*, the employer must prove by "the best available scientific evidence" that: 1) significant risks of fetal harm would result from the mother's exposure, 2) the risk is substantially confined to female and not male workers, and 3) the FPP is effective in significantly reducing the risk. The employer's subjective motivation and good faith belief that the FPP is necessary and effective is insufficient to prove necessity or effectiveness. The essentially scientific nature of these issues requires opinion evidence of qualified experts in the relevant scientific fields. To establish the requisite degree of risk, the employer need not prove the existence of a general consensus within the qualified scientific community. However, the employer must show that within that community there is a considerable body of opinion that significant risk exists and that the risk is substantially confined to women workers, so that an employer could not responsibly fail to act on the assumption that this opinion might be the accurate one.[63] Once the employer has established the business necessity defense, the plaintiff may nevertheless prevail by proving that there are "acceptable alternative policies or practices" that would better accomplish the business purpose, or accomplish it equally with less disparate impact.[64] Furthermore, pretextual policies are still unlawful.

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[53] U.S. Department of Health and Human Services, National Center for Health Statistics, Advance Report of Final Natality Statistics, 1981, *Monthly Vital Statistics Report*, vol. 32, No. 9, supp., DHHS Publication No. (PHS) 84-1120 (December 1983).

[54] 697 F.2d at 1176.

[55] Wright v. Olin Corp., 585 F. Supp. 1447 (W.D.N.C.), *vacated*, No. 84-1276 (4th Cir. Aug. 31, 1984).

[56] 697 F.2d at 1187.

[57] *Id*. at 1185 n.21.

[58] *Id*. at 1188.

[59] *Id*. at 1188, quoting the landmark discrimination case, Dothard v. Rawlinson, 433 U.S. 321, 335 (1977).

[60] *E.g.*, Burwell v. Eastern Air Lines, Inc., 633 F.2d 361 (4th Cir. 1980) (en banc), *cert. denied*, 450 U.S. 965 (1981), which held that airline passenger safety justifies a policy of mandatory leave for pregnant stewardesses.

[61] 697 F.2d at 1189.

[62] *Id. at 1190.*

Under the *Olin* analysis, such rebutting evidence may have either of two effects, both resulting in employer liability, but with possibly different consequences. If the plaintiff shows the existence of an acceptable alternative, she would be entitled to a judgment that vindicates (in both injunctive and monetary award aspects) the plaintiff's rights as they would exist under the acceptable alternative policy. On the other hand, if the plaintiff can prove that the acceptable alternatives were not implemented because of the employer's *discriminatory intent*, the plaintiff would be entitled to a judgment wholly freed of any restrictions due to the alternative policy.

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[63] *Id.* at 1191.

[64] *Id.*

CASE STUDY: AMERICAN CYANAMID'S FETAL PROTECTION POLICY

In January 1978, the American Cyanamid Co. announced that all fertile women would be removed from exposure to certain toxic substances at its Willow Island, West Virginia, plant. This policy, implemented in October 1978, required that women of childbearing capacity not be assigned to jobs, or allowed to bid on jobs, that involved exposure to substances the company believed were harmful to fetuses. As a result of this fetal protection policy (FPP), two women workers were transferred to janitorial jobs, while several other women underwent surgical sterilization because they feared they would lose their jobs. In early 1980, these women and others affected by the FPP filed suit against Cyanamid, claiming that the company's fetal protection policy constituted sex discrimination in violation of Title VII. After 3½ years of pretrial proceedings and shortly before the trial was to begin, the case was resolved by an offer of judgment for $200,000 plus costs and attorneys' fees, pursuant to Federal Rule of Civil Procedure 68.[65] There was no admission of liability by the company.

This case study describes how one firm, the American Cyanamid Co., became suspicious that its workers might be exposed to reproductive health hazards and describes the steps leading to the announcement of a fetal protection policy excluding women from some work assignments. The chronology of events suggests that the company initiated its exclusionary policy with little scientific justification and little sensitivity to the needs of its workers, though to its credit, Cyanamid responded to some of the OSHA and labor union criticisms of the policy.

Since a number of major corporations have implemented, or are considering, similar exclusionary policies, the Cyanamid story suggests that industry needs to develop greater sensitivity and education on the reproductive hazards issue. While it is not clear that the Cyanamid case is representative of these policies, it is illustrative of how one major corporation attempted to deal with the possible risks caused by potential reproductive health hazards in the chemical workplace. Appendix 8A describes the policies of some other large companies and hospitals.

This description of events leading to the implementation of the fetal protection policy is based on portions of sworn deposition testimony taken by counsel for the plaintiffs of a physician who served as Cyanamid's Corporate Medical Director during the relevant period. Cyanamid has reviewed a draft of this chapter and has presented some of its comments in the critique that follows this case study.

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[65] Rule 68 allows a Federal court defendant to offer to allow a judgment to be taken against him for a specified amount of money. If the plaintiff fails to accept the offer and wins a judgment that is less favorable than the defendant's offer, the plaintiff must pay the costs incurred after the making of the offer.

At the time the FPP was developed, Cyanamid's central medical department reported to the personnel director and was composed of three programs: toxicology, industrial hygiene, and employee health. The toxicology group was composed of toxicologists who worked in a specialized laboratory performing animal studies of the effects of chemicals used in Cyanamid plants. The industrial hygiene group, composed of five centrally located industrial hygienists as well as resident hygienists at three plants, was charged with conducting industrial hygiene surveys of every Cyanamid plant in the United States and Canada. Plants were surveyed at least annually, although larger plants and those with complex product mixes were surveyed as frequently as every month. As a result of these surveys, and in conjunction with the central medical department, the industrial hygienists set permissible exposure limits for chemicals encountered at Cyanamid plants. The corporate medical director was the only person with the authority to change these permissible exposure levels. The employee health group was composed of: 1) 2 centrally located physicians, who were responsible for implementing the employee health program throughout the company, 2) 15 medical offices located at various Cyanamid plants, and 3) approximately 130 "fee for service" physicians who worked for the company as needed. The medical officers reported to the corporate medical department informally as needed and on a formal basis once each month. They reported all medically related activities during the previous month, including deaths, serious accidents, lesser but recurring accidents (e.g., eye irritation), personnel changes, physical examinations, and evaluations of employee exposure to toxic substances. Cumulative reports were also made to the central medical department on an annual basis.

In 1975, the corporate medical director first perceived a potential problem for women of childbearing capacity who worked with toxic chemicals. Although he did not know the magnitude of the problem, he believed that it was to be one of increasing importance because more and more women were bidding on jobs in heavy chemical areas. He was concerned that this change in employment patterns might pose a risk to the embryos and fetuses of employees. The medical director believed the risk to employees from possible reproductive health hazards to be greater than the risk from suspected carcinogens, since exposure to suspected carcinogens was either eliminated (through substitution of nonsuspect chemicals) or reduced significantly. He defined the reproductive health hazards problem as one of embryofetotoxicity (toxic effects on the embryo or fetus) due to the exposure of either parent to hazardous chemicals. He considered embryofetotoxicity to have four components: direct toxicity to the fetus, mutagenicity, teratogenicity, and transplacental carcinogenicity. Such a definition excludes negative reproductive outcomes such as infertility and sterility.

The medical director claims he was initially concerned with all embryofetotoxic effects of chemicals used by Cyanamid but later decided to focus exclusively on the potential adverse effects to the fetus transmitted through the mother. The medical director stated the reasons for this change in focus to be because of his "professional judgment" that there was a much more compelling body of evidence concerning embryofetotoxicity as mediated through the mother than through the father.

Prior to announcing the FPP, the medical director had considered applying the policy only to women who were pregnant or planning pregnancies, but rejected this approach as being impractical because of his belief that most women are unaware of their pregnancies at the early stages. However, when rejecting this approach, the medical director had no specific information suggesting that any of Cyanamid's chemicals had an impact on the embryo during the first 3 months of pregnancy.

In August 1976, after much discussion within the central medical department and approval by the personnel officer, the medical officer circulated a memo to senior management containing an FPP applying only to female production workers. The FPP prohibited "female employees in the childbearing age (considered in industry to be 16 to 55 years)" from working in production jobs where they would be exposed to any of 29 chemicals listed in the policy memo, regardless of the level of exposure. The medical director expected the policy to be effective immediately and to affect the jobs of 25 to 50 female Cyanamid employees.

As of the time the FPP was proposed, no assessment had been made of the degree of risk to the offspring of either male or female employees. Although the medical director was unable to quantify the risk of a woman worker bearing a child damaged by workplace exposure, his professional judgment led him to believe that such an outcome was a "likely possibility." Although the medical director's assessment of the likelihood of harm included consideration of exposure levels, he felt that he could not determine with certainty what a safe exposure level for an embryo/fetus would be, given the greater susceptibility of an embryo/fetus. For this reason, exposures at any level were prohibited.

In addition, the statement of the medical director indicates:

1. The medical director had never instructed plant physicians to inquire about fertility or reproduction problems among production workers.
2. The company had never conducted or commissioned an epidemiological or other study designed to determine whether any employees had suffered from any form of reproductive toxicity.
3. No organized collection of sperm samples of male employees was ever proposed or conducted.
4. No studies were made to determine whether Cyanamid employees or their children had chromosomal abnormalities.
5. The medical director had never issued any kind of instructions to plant physicians about counseling or treating employees who were exposed to reproductive toxins.
6. The medical director was not aware of any cases in which an employee was reproductively harmed or a child, fetus, or embryo was affected as a result of workplace exposures at a Cyanamid plant.
7. No studies were performed on the childbearing patterns of the production force.
8. Although members of the Central Medical Department had looked up certain articles on reproductive toxins, they did not perform a literature search or research project for internal discussion.

The list of 29 substances was "compiled as a result of a quick review of computer sheets." No animal studies were performed. The medical director knew the effects of lead on an embryo or fetus resulting from maternal exposure from the writings of several epidemiologists, but had no specific information as to whether any of the other 28 chemicals were embryofetotoxins. The selection of these substances was based on volume of use, toxicity to adults, and a professional judgment that any substance that was highly toxic to an adult might be even more toxic to an embryo or fetus. The medical director identified nine of the substances as being suspected carcinogens and, in fact, three of these were placed on the list solely because of their carcinogenic (as opposed to toxic) potential. For these nine chemicals, the medical director was more concerned with their potential effects on an embryo or fetus than with potential carcinogenicity in adult workers. For the three chemicals that were placed on the list due to their potential carcinogenicity, he was concerned that the embryo or fetus might either develop cancer in and of itself or contract cancer due to metastasis of chemically induced cancer in the mother. No materials were prepared addressing the possibility of such alternatives to the FPP as engineering controls, substitution of chemicals, the use of personal protective equipment, or job rotation.

The FPP applied only to female production workers. Research personnel were exempted from policy coverage because the medical director believed that laboratory hazards were better controlled than hazards in production facilities. However, the medical director had no knowledge of what kinds of substances female research personnel were exposed to or whether these employees used protective equipment.

The original policy was circulated but not implemented. Some Cyanamid managers expressed concern as to whether there was in fact a significant danger to women, whether research personnel should be exempt, and whether such a sweeping company-wide policy should be implemented without the advice of the company's Executive Committee.

In September 1976, the Executive Committee held up implementation of the policy and asked for additional information. The personnel director sent a confidential memo to the presidents of all Cyanamid divisions, listing the 29 chemicals and asking that the divisions indicate how many male and female production workers were exposed to each chemical. In addition, the female employees were to be listed by name, age, department, and frequency of exposure. The survey responses led the Central Medical Department to believe that, in "many instances," the female employees' exposure was not significant.

Nevertheless, guidelines for implementing the FPP were circulated in December 1976. They continued to propose prohibiting any exposure to women workers, even on an occasional basis. The guidelines did, however, revise the class of women affected. "Childbearing potential" was redefined as occurring before the age of 50, rather than 55. This change resulted from discussions between the medical director and his staff concerning the unlikelihood that a woman would conceive past the age of 50. (The possibility of lowering the maximum age to 45 had been considered but rejected because the medical director believed that "any numbers of pregnancies" occur between the ages of 45 and 50. However, the medical director stated that he was unaware of the proportion of pregnancies that occur between those ages. As noted in chapter 7, only 1 of every 5,000 women aged 45 to 49 gives birth each year.) In addition, the guidelines suggested that a 6-month period be allowed for voluntary reassignment of female employees. The original FPP provided no such transition period.

Throughout the 1-year period beginning with the announcement of the original version of the FPP, the medical office's research into the potential risks and hazards associated with Cyanamid remained at a low level. The medical librarian was asked to review any new publications relevant to the FPP, but the medical director was unable to recall any specific occasions on which the medical librarian in fact forwarded an article to him. No specific research was performed, except for a list of references compiled by the associate medical director. No research was undertaken to address the possibility of alternatives to the FPP.

In September 1977, the Executive Committee approved a modified fetal protection policy, subject to the concurrence of the legal and insurance departments. The new policy was similar to the first policy. Childbearing age was defined in the new FPP as 16 to 50 instead of 16 to 55. The language in the new policy was milder than in the original FPP; for example, while the first memo stated that certain chemical and physical agents "have the capacity to cause developmental defects," the new policy stated that these substances "may" have this capacity. The December 1976 guidelines were incorporated into the new FPP. Like the original policy, the FPP distributed in September 1977 was limited to female production workers, prohibited any exposure whatsoever to the 29 substances, and was intended to be effective immediately. This policy was announced but not implemented.

Shortly after the announcement of the new policy, several industrial hygienists and an associate medical director suggested that exposure limitations be substituted for the exposure prohibition. They felt that it would be inappropriate to prohibit employees from experiencing workplace exposure to substances to which they were exposed in the environment. In October 1977, the medical office issued a set of maximum permissible exposures for women employees who were exposed to any chemicals on the list. The maximum permissible exposures for fertile women between 16 and 50 years of age were set at a fraction of the maximum permissible exposures recommended for adults by the American Conference of Governmental Industrial Hygienists. (This fraction was determined by the industrial hygienists, and the medical director did not know how the fraction was derived.) The substitution of exposure limits for the total exclusion of fertile women had little practical effect, however, as the maximum exposures were so low as to require the exclusion of most women working with most of the chemicals. In a letter to the medical director of Western Electric Co., Cyanamid's medical director stated that "we have not determined a safe level of exposure but have arbitrarily taken fractions of existent threshold limit values and employ these as threshold limit values for fertile females."

In early November 1977, representatives of Cyanamid, OSHA, and the National Institute for Occupational Safety and Health (NIOSH) met to discuss Cyanamid's policy on female production workers. The meeting was held at OSHA's request after the United Steelworkers of America filed a complaint to an OSHA area office. OSHA and NIOSH expressed three major concerns: 1) the lack of scientific data to support the inclusion of the 29 listed materials, 2) the possibility that women would be eliminated from the chemical workplace, and 3) the possibility that several companies would each set their own permissible exposure levels below the OSHA levels. At the meeting, the medical director stated that Cyanamid had not conducted studies to generate new data about the effects of the chemicals, but had relied on "extensive literature research and experience" to arrive at professional judgments and that 12 months of time were spent on this review. When asked whether Cyanamid had considered a policy addressing the potential effects of chemicals on male reproductive function, the medical director replied that he was not aware of any information concerning adverse effects on male reproductive function. When asked whether Cyanamid planned to conduct research aimed at supporting its FPP, the medical director stated that a $40,000 project had been approved to study the teratogenic effects of acrylamide, one of the 29 substances, and that additional research activities were expected. When a NIOSH representative pointed out that the NIOSH Criteria Document on acrylamide stated that no teratogenic effects were known, the medical director indicated that he was aware of this, having served as a review consultant for the document. (Acrylamide was the only one of the 29 substances to be tested by Cyanamid. It was selected for study because of labor relations problems at one of Cyanamid's plants resulting from the FPP's inclusion of acrylamide. As a result of the study, acrylamide was removed from the list.)

Also in November 1977, the medical department issued a second set of Permissible Exposure Limits (PELs) which contained ceiling limit values as well as time-weighted average values and provided time-weighted average values for the different physical states of the chemicals. In every instance, the ceiling limit values were three times greater than the 8-hour time-weighted average. No comparisons were made between the values set and the actual exposure levels in the company's plants.

In late November 1977, the medical director sent a memo to the personnel director concerning guidelines for fertile female employees who worked in Cyanamid's laboratories. The memo stated that if workers followed existing laboratory rules, exposures would be below the PELs established by the medical department.

In December 1977, the medical director wrote a letter to the assistant corporate medical director at E. I. du Pont de Nemours & Co., in which he stated that the PELs "were arrived at quite arbitrarily and really constitute an educated professional guess rather than anything that we could document on the basis of clinical or laboratory experience."

Although the medical director excluded fertile female production workers from exposure to the 29 chemicals with virtually no data to support this policy, he stated that he was unwilling to exclude fertile men in the absence of "epidemiological studies indicating that the compound was indeed a human mutagen." He would not be persuaded by animal studies showing evidence of a chemical's mutagenic effect on sperm and claims that "the only meaningful information that [he] would accept is epidemiological information."

The fetal protection policy was announced to workers, though not actually implemented, at some Cyanamid plants in late 1977 and early 1978. The corporate FPP was silent as to whether implementation was to be on a departmental or job-by-job basis. At the Willow Island plant, women were informed in January that, beginning on May 1, those under 50 who were not surgically sterilized would be excluded from 8 of the plant's 10 departments. No mention was made of PELs and no monitoring had been done to determine whether exposure levels for all of the jobs in the exclusionary departments were in excess of the PELs established by the corporate medical department. Employees were informed that fertile women would only be employed in the remaining two departments or in janitorial positions. Positions in these departments would be subject to the departments' personnel needs and wages. In most cases, women transferring out of an exclusionary department would receive lower wages in the new department. There was no assurance that a sufficient number of jobs would exist in unaffected departments to accommodate all women displaced by the FPP, in which case women were expected to be laid off.

At a later time, Cyanamid reconsidered the exclusion of female laboratory workers from the bounds of the FPP after receiving reports from industrial hygienists that not all laboratory workers were observing the cautionary guidelines. Fertile female laboratory workers were therefore made subject to the FPP, but the policy was never in fact enforced for laboratory workers.

In early 1978, the supervisor of industrial relations at Willow Island asked the medical director whether the FPP should be implemented on a departmental or an individual basis. The medical director informed him that the policy had always been to consider each individual job rather than to require exclusion by department. However, the medical director did not believe it was necessary to make this clarification on a corporate level because he believed that consideration by individual job could be inferred from the written policy. The medical director interpreted the Willow Island announcement as excluding fertile women on a job-by-job basis rather than on a departmental basis, even though the announcement stated that:

> . . . [t]he Departments in which female production employees with childbearing potential will not be permitted to work after May 1, 1978 are as follows. . . . These female employees are encouraged to submit requests for transfer, in accordance with the [union] contract, to the following Departments. . . . These are the only Departments where female employees of childbearing potential will be permitted to work after May 1, 1978. Those female employees of childbearing potential who remain in the [exclusionary] Departments . . . will be subject to reassignment or to layoff . . .

In April 1978, the Office of the Chairman (which replaced the executive committee) announced that implementation of the FPP was to be further delayed until July. The delay was based on concerns, expressed by both union and management officials, as to the magnitude of the risk and the policy. In June 1978, the Office of the Chairman decided to defer implementation of the FPP until September 1 and announced that prior to that date the newly formed Occupational Exposure Review Committee (OERC) would review and appraise the scientific basis for the PELs and FPP and report back to the Office of the Chairman. Although the medical director was satisfied that he had sufficient information to support the PELs and the FPP, he agreed with the formation of the OERC "in view of the fact that the company had decided that they wanted documentation of a scientific nature" and the use of "professional judgment" should play a lesser role. The OERC's mandate was to review the scientific literature concerning the list of 29 compounds, analyze the documentation for the PELs established by the medical department, and determine whether any of the compounds should be deleted from the list or subject to different PELs. The medical director stated that the OERC had authority to inquire into the effects of chemical exposures on male reproduction and the children of male workers, as well as the effects on female workers and their children.

The OERC review resulted in exposure limitations (and exclusion of fertile female workers who would be exposed in excess of these levels) for only six compounds: lead, diamox, hydrazine sulfate, hydrazine hydrate, methotrexate, and thiotepa. The new FPP was to apply to women between the ages of 16 and 50, both production and laboratory workers, who were not proven incapable of childbearing. Women whose job assignments resulted in exposure in excess of the PELs would not be terminated but given alternate assignments and wage rate retention for a "reasonable period of time and under reasonable conditions." With the reduced list, it appeared that the FPP's impact would be limited to eight female employees at the Willow Island plant. Several women there already had themselves surgically sterilized in response to the original announcement in January 1978, before the new FPP was finally implemented at Willow Island in the fall of 1978. In February 1979, the FPP was again revised, with diamox deleted from the list. In late 1979, the lead pigment department was shut down by Cyanamid, a year after the FPP was announced.

In 1979, OSHA issued a citation claiming that Cyanamid's fetal protection policy violated section 5(a)(1) of the general duty clause of the Occupational Safety and Health Act of 1970. (See discussion in chapter 7.) OSHA argued that the general duty clause requirement that employers provide employment free of "recognized hazards" prohibited any condition of employment that could ultimately result in reduced functional capacity, including FPPs that might result in some employees undergoing surgical sterilization. OSHA's citation was struck down by the Occupational Safety and Health Review Commission, which ruled that Congress did not intend "recognized hazards" to include policies that might encourage sterilization. The Commission's decision was affirmed by the District of Columbia Court of Appeals.

According to a reconstruction of the events of 1978 by the U.S. Court of Appeals for the District of Columbia:

> In January and February of 1978, Glen Mercer, the plant Director of Industrial Relations, conducted a series of meetings for small groups of the Willow Island plant's female employees.
>
> At these meetings, Mercer informed the women that hundreds of chemicals used at the plant were harmful to fetuses and that, consequently, the company had decided to exclude women of "childbearing capacity" from all departments of the plant where such chemicals were used.
>
> Mercer further declared that the company would deem any woman between the ages of 16 and 50 to be of childbearing capacity unless she presented proof that she had been surgically sterilized.
>
> A company doctor and nurse accompanied Mercer to these meetings and addressed the women. They explained to the women that "buttonhole surgery" was simple and that it could be obtained locally in several places. The women were also told that the company's medical insurance would pay for the procedure, and that sick leave would be provided to those undergoing the surgery.
>
> Mercer told the women that once the fetal protection policy was fully implemented, the plant would have only about seven jobs for fertile women in the entire facility. Approximately 30 women were then employed at the plant.
>
> Apart from the women who obtained those seven positions, Mercer said that female employees who failed to undergo surgical sterilization by May 1, 1978 would be terminated. The company extended the May 1 deadline several times. In September 1978, the company informed the women of changes in its policy. The deadline had been extended to October 2, 1978, the inorganic pigments department was the only department affected, and the only material covered by the policy was lead. . . .
>
> Between February and July 1978, five women employed in the inorganic pigments department underwent surgical sterilization at a hospital not connected with the company. Two women in that department did not choose sterilization. The company transferred them into other departments and, after 90 days, lowered their rate of pay to correspond to the rates characteristic of their new jobs."[66]

Would Cyanamid have acted differently had it realized that its fetal protection policies would provoke a lawsuit? Would the company have acted differently if application of a FPP had resulted in requiring men to be sterilized to keep their jobs? The $200,000[67] offer of judgment may have been less expensive than either a more comprehensive research effort or the institution of engineering controls to prevent potentially hazardous exposures. It may also have been less expensive than a lawsuit by the defective child of an exposed worker. It is not clear whether the economic disincentive of facing a sex discrimination lawsuit is sufficient to alter company policies.

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[66] Oil, Chemical, and Atomic Workers International Union v. American Cyanamid Co., 741 F.2d 444 (D.C. Cir. 1984).

[67] However, the cost of defending such a case may be significant. A defendant may also have to pay the plaintiff's costs. In the *Cyanamid* case, a substantial claim for attorneys' fees is still pending.

These questions and those that follow are intended to be generally illustrative and to raise issues, not to impugn the motives of a specific company.

The evolution of American Cyanamid's FPP raises a number of policy questions about corporate decisionmaking concerning potential reproductive health hazards in the workplace. Should employers seeking to identify reproductive health hazards and develop a protective health policy be required to make these decisions in a certain way? If so, what should be required? To what degree should an employer be permitted to err on the side of caution? Should this discretion vary, depending on either the severity or permanence of the potential health effect? Should this discretion vary with the economic burden it places on employees? If the existence of a reproductive hazard is suspected, should a company have the right to modify the work force rather than modifying the workplace? Should limits be placed on the extent to which a company can exclude women?

What constitutes sufficient scientific evidence to establish or rebut hazardousness and unacceptable riskiness for the purpose of implementing a protective policy? In the absence of sufficient scientific evidence regarding hazardousness, what weight should be given to professional medical judgment? If scientific evidence establishing or rebutting hazardousness is available, should professional medical judgment be an acceptable substitute? Should professional medical judgment be sufficient to establish the existence of a reproductive hazard for the purpose of implementing a protective policy that places the economic burden on the worker rather than the employer? Should professional medical judgment be sufficient to rebut a hazard for the purpose of avoiding a protective policy?

Although the courts have tentatively answered a few of these questions (see chapter 10), many of them remain unresolved. As long as these questions have no clear-cut answers, companies may continue to institute exclusionary policies that are discriminatory. Or they may not control exposure to reproductive health hazards in their workplaces.

OTA's Note: *OTA requested comments, criticism, and clarification from American Cyanamid on a draft of this case study. Approximately half of the company's comments resulted in revisions that are reflected in the foregoing material. The remainder are reprinted below.*

American Cyanamid Co. Response

The following are limited comments of American Cyanamid as requested by the Office of Technology Assessment (OTA) on its case study of Cyanamid's fetal protection policy (FPP). The OTA draft is based solely on deposition testimony of the retired corporate medical director of Cyanamid. As such, it does not reflect the involvement of other key Cyanamid personnel directly involved in the development of the policy and is limited to subjects that plaintiffs' counsel chose to pursue in questioning. OTA requested Cyanamid to limit its comments to a specific and very short critique of the draft, and it has attempted to meet that requirement. However, the company does not intend these comments to be interpreted as reflecting its agreement with other statements in the draft. To the maximum extent, the comments track the sequence of topics covered in the draft case study:

- The draft omits some critical events. The FPP was implemented in a form substantially revised from that announced in January 1978, after extensive consideration by the Occupational Exposure Review Committee (OERC), composed of Cyanamid's top medical and scientific professionals, and top management. Moreover, as ultimately put into effect in October, only employees working with one substance (lead) and in one department were affected. No employee lost a job as a result of the FPP. Of the two employees who were required to transfer from production to janitorial positions, one transferred at the same pay rate; the other had her prior wage rate retained on transfer. Furthermore, those two employees had opportunities to transfer back into production positions. Indeed, one employee declined an offer to transfer back into a production position while the other requested permanent assignment to the Janitors Department.

- The draft should also be revised to reflect that, when announced, both health professionals and management at the plant expressly discouraged female workers from undergoing sterilization procedures.
- The draft incorrectly suggests that Cyanamid did not consider infertility, sterility, or potential effects on the offspring mediated through paternal, as opposed to maternal, exposure to workplace chemicals. Cyanamid did in fact consider all those risks. However, it considered infertility and sterility to be adult, rather than fetal health risks, and, thus, protected via its existing health program. With respect to risks via paternal exposure, both Dr. Clyne and OERC, in its review of the FPP, continued to consider all available evidence of male-mediated risks.
- Contrary to the implication in the draft, Cyanamid considered the proper scope of the FPP throughout 1976-79. Whether the policy could be restricted to pregnant women was a specific item of discussion at the OERC in the summer of 1978, before the policy was implemented, as well as a subject of concern for Dr. Clyne in 1976.
- The draft concentrates on events that took place prior to September 1977, and, therefore, fails to put the development of the FPP in proper perspective. It particularly fails to discuss the critical importance of the OERC in developing and refining the policy. The draft should make clear the following sequence of events. Dr. Clyne circulated a statement of his proposed FPP to senior management in August 1976, but the Executive Committee directed that no further action be taken to implement the policy. The Executive Committee did not approve in principle the FPP until September 1977, and even then, implementation was postponed pending further study. In June 1978, top management created the OERC, which functioned as a peer review panel, to reexamine the scientific documentation of risks to the fetus for the 29 substances then subjected to the proposed policy. The revised policy (narrowed to six chemicals) received management approval in August 1978.
- The draft, by focusing only on the very early stages of the policy, misleadingly suggests that chemicals were included in a haphazard basis. The deposition makes clear that, in selecting the substances, Dr. Clyne and his staff proceeded cautiously and on the basis of their very extensive experience in the occupational health, toxicology, and industrial hygiene fields. All were familiar with the scientific literature regarding toxicity of chemicals in use at Cyanamid and employed the widely accepted convention that the rapidly differentiating tissue and speed of development of the fetus would enhance its susceptibility to certain substances known to be toxic to adults. Finally, the OERC's detailed review of the scientific literature in 1978 should be acknowledged. The OERC's consensus conclusion from that continued examination was that six (later five) substances did require special exposure standards for fertile women. As to the others, the draft should make clear that OERC did not dismiss them as not toxic to the fetus, but rather concluded only that the scientific documentation of risk was not such that company action was required.
- Contrary to the impression created by the draft, the company's corporate medical staff had given considerable attention to the "exposure limit" issue prior to the initial issuance of the policy in September 1977. The staff adopted a "zero exposure" standard for the substances covered by the policy because they felt that a very conservative approach was justified on the issue of fetal health, particularly given their knowledge that the exposure level at which no effects on the fetus would occur was uncertain for these substances. The OTA draft also incorrectly suggests that the company's subsequent adoption of exposure limits in order to make its approach to fetal health as consistent as possible with its approach to adult health "had little practical effect, as the PELs were so low as to require the exclusion of most women working with most chemicals." These limits formed the basis for Willow Island's job-by-job approach to the policy's implementation, which carefully limited the number of posi-

tions to be covered by the policy. Moreover, the OTA draft fails to recognize that the policy, as ultimately implemented at Willow Island in October 1978, covered only one chemical at that plant and affected only one department, requiring the transfer of only two employees.

- The draft also takes out of context Dr. Clyne's use of the word "arbitrarily" in describing to a colleague the methodology used for setting policy exposure limits in October 1977. "Arbitrarily" merely signified that the company's medical staff had not attempted to quantify scientifically the actual "no effect" level below which there would not be a risk in the fetus. Dr. Clyne and his staff had employed professional judgment in selecting limits, lower than the permissible adult level, that they believed would be protective of fetal health. Contrary to the implication in the draft, it would have been inappropriate for the staff, in setting these limits, to compare them with actual exposure levels in the plants.
- The draft presents an incomplete account of the fall 1977 meeting OSHA and NIOSH had requested with the company to discuss its policy. Most importantly, it ignores the OSHA representative's commendation to the company for its efforts to provide a safer workplace than required by OSHA standards.
- The draft is misleading in asserting that no materials were prepared or research done to address the possibility of alternatives to the exclusion of women of childbearing capacity, such as engineering controls, personal protective equipment, or job rotation. First, it was the role of the company's operating divisions, not the corporate medical staff, to address the "operational alternatives" issues. Secondly, the company had conducted studies that allowed the operating divisions to assess the alternatives issue without additional research. The company's industrial hygienists had studied engineering controls in the Lead Pigments Department at Willow Island in 1972 and 1977. Engineering controls installed as a result of the 1972 study were found to have had little impact on reduction of lead-in-air levels. The company also had considered the reliability of various respirators and had concluded, consistent with the literature in the respirator field, that factors such as the fit of the respirator on the wearer's face significantly reduced the reliability of this alternative. Finally, the OERC-revised policy required consideration of alternatives in implementing the policy. The Organic Chemicals Division gave specific consideration to engineering controls, respirators, and job rotation in the fall of 1978 and determined that there were no feasible alternatives to the exclusion of women of childbearing capacity from the Lead Pigments Department at Willow Island.
- Cyanamid strongly disagrees with the draft's suggestion that the company might have preferred the cost of Title VII litigation to the costs necessary to engage in more comprehensive research, to develop better engineering controls, or to resolve a lawsuit involving a defective child. This paragraph should be deleted. First, there was not the slightest suggestion in the testimony or documents that the express purpose of the policy was not to protect the fetus. The policy was not adopted because of concern for potential financial liability or as a substitute for more expensive exposure controls. Indeed, the risks of injury to the fetus from chemical exposure cannot be calculated in financial terms. Cyanamid's expenditures to limit chemical exposures in the workplace are very substantial and demonstrate its longstanding commitment to this goal. The FPP was a further step in the fulfillment of that safety objective, not a convenient substitute for it.

CONCLUSION

The spectrum of employers instituting or considering fetal protection policies ranges from large chemical and automobile manufacturers to small community hospitals. Although it is impossible to determine how many companies have either written or unwritten exclusionary policies, at least 15 of the Fortune 500 as well as numerous hospitals are reported to exclude fertile and/or pregnant women from some jobs.

There is tremendous diversity in company exclusionary policies. Some of these policies are strongly grounded in epidemiological and toxicological research findings with respect to particular substances, while others are more speculative about potential reproductive health hazards. Some policies are carefully written and documented, while others are unwritten, making them more flexible but also more ambiguous. In large manufacturing companies, policies are generally announced to employees and their unions prior to implementation, while smaller organizations appear to formulate and apply policies as a perceived problem arises. Some policies recognize that a fetal hazard may be mediated through either the male or female workers, while others apply only to women.

In some cases, these policies have faced court challenges on grounds of sex discrimination in violation of Federal law. Title VII of the Civil Rights Act of 1964 prohibits employment discrimination on the basis of sex, while the Pregnancy Discrimination Act of 1978, an amendment to Title VII, specifically forbids discrimination on the basis of pregnancy, childbirth, or related medical conditions. The law requires that women affected by these conditions be treated the same for all employment purposes as others not so affected but similar in their ability or inability to work.

While many of these cases are apparently settled out of court, some have been adjudicated and three have been decided by the Federal courts of appeals in the Fourth, Fifth, and Eleventh Circuits. All three courts have held that the exclusion of fertile or pregnant women constitutes illegal sex discrimination under some circumstances. Although the three courts used different approaches, the following general principles can be extracted from these cases:

- A fetal protection policy (FPP) that applies only to women is presumptively discriminatory. That is, the mere existence of an FPP will create Title VII liability for the employer in the absence of strongly supportive scientific evidence.
- To overcome the presumption of discrimination, the employer must be able to prove that the body of scientific evidence supports legal findings that: 1) exposure at the level encountered in the workplace involves a significant risk of harm to the unborn children of women workers, 2) exposure at the level encountered in the workplace does not involve a similar risk of harm to the future offspring of male employees, and 3) the FPP is effective in significantly reducing the risk. An employer's subjective but scientifically unsupportable belief in the necessity of the policy is insufficient to defend it.
- If the employer proves both points embryo/fetal risk through maternal exposure and lack of embryo/fetal risk through paternal exposure), the plaintiff may nevertheless prevail by proving that an acceptable alternative policy would promote embryo/fetal health at least as well with a less adverse impact on one sex or by showing that the FPP is a pretext for discrimination.

TECHNICAL NOTE 8-1: LITIGATION OF SEX DISCRIMINATION CASES

Discriminatory Treatment

The Supreme Court established the framework by which the factual issues are resolved in a Title VII case of discriminatory treatment.[68] The most notable feature of this framework is that the burden of proof shifts back and forth between the plaintiff-employee-applicant and the defendant-employer. The framework is applicable to cases of claimed discrimination in hiring, promoting, and firing.

The plaintiff has the initial burden of proof to establish a prima facie case of disparate treatment. A prima facie sex discrimination case is established by showing that the plaintiff: 1) is female, 2) applied for a position for which the employer was seeking applicants, 3) was qualified to perform the job, 4) was denied the job, and 5) the employer hired a male or continued to seek applicants for the job. A plaintiff's failure to establish all five facts will generally result in a judgment in favor of the employer.

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[68] McDonnell Douglas Corp. v. Green, 411 U.S. 792 (1973).

If the plaintiff establishes a prima facie case, however, the burden of proof shifts to the employer. The plaintiff is entitled to win as a matter of law unless the employer proves either that sex is a bona fide occupational qualification (BFOQ) or that there are "legitimate and nondiscriminatory reasons" for the plaintiff's rejection. Examples of legitimate reasons for rejecting the plaintiff include inadequate qualifications, experience, seniority, and performance. An employer's failure to prove legitimate reasons for failing to hire the plaintiff will result in a judgment in the plaintiff's favor.

If the employer proves legitimate reasons for refusing to hire the plaintiff, the ball is back in the plaintiff's court. To prevail, the plaintiff must prove that the employer's apparently legitimate reasons were merely a pretext for an illegal discriminatory motive. A plaintiff could show such a pretext by demonstrating, for example, that the employer's asserted criteria were not applied uniformly to all applicants, that the employer had a history of discriminating against women, or that the employer made work assignments in such a way as to cause the plaintiff's poor performance. If the plaintiff produces evidence of a pretext for discrimination, the employer may produce his or her own evidence in response. The court then examines all of the evidence to make a determination as to whether the employer's rejection of the plaintiff was motivated by improper purposes or based on the legitimate reasons presented.

Disparate Impact

There are fewer steps involved in litigation of disparate impact cases. First, the employee or applicant must prove that an employer's specific employment policy or general employment practices have a disproportionately adverse impact on a protected class; she need not prove discriminatory intent. If the plaintiff fails to demonstrate an adverse impact, the employer wins. If the adverse impact is demonstrated, the employer must prove that the policy is a business necessity. If the employer fails to demonstrate a business necessity, the plaintiff wins.

Despite the seeming simplicity of this formula, proving disparate impact is often extremely complex. One method uses *applicant flow* data. Under guidelines established by the EEOC, a selection process will normally be considered to have a discriminatory impact if the selection rate for any race, sex, or ethnic group is less than 80 percent of the rate for the group with the highest rate.[69] For example, if 100 women apply and 20 are hired, the female selection rate is 20 percent. If 150 men apply and 45 are hired, the male selection rate is 30 percent. Since the female selection rate is only 67 percent of the male selection rate, the hiring policy would generally be considered to have a discriminatory impact. If at least 24 women had been hired, the policy would generally be considered nondiscriminatory.

There may be problems with using applicant flow data and the 80 percent rule, however. Selecting an appropriate sample for applicant flow data comparison is often extremely difficult. For example, in a lawsuit by a black female applicant for a managerial engineering job, a court must make two initial determinations: should it look at the company's record of hiring women, blacks, black women, or minority women, and should it look at these applicants for all professional jobs, for engineering jobs, or for managerial jobs? Often, these determinations will dictate whether the employment policy meets the 80 percent requirement. Furthermore, the 80 percent rule is far from absolute. Smaller differences in selection rate may nevertheless constitute adverse impact where they are significant in both statistical and practical terms, or where the employer's actions (or history of discriminatory practices) have discouraged applicants disproportionately on grounds of race, sex, or ethnic group. Greater differences in selection rate may not constitute adverse impact where the rates were derived from a statistically insignificant applicant pool, or where special recruiting programs cause the pool of minority or female applicants to be atypical of the normal pool of applicants from that group. If an applicant pool is too small to be statistically significant, evidence may be introduced concerning the impact of the policy over a longer period of time or concerning the impact that the selection procedure had when used in the same manner in similar circumstances elsewhere. When time-frame analysis must be done, the question arises as to which of the infinite number of possible time frames is most appropriate for analysis. This is sometimes complicated by the fact that employment policies change over time so that no time frame contains all of the employment policies challenged by the plaintiff. If a comparison is made with similar policies used in similar circumstances by other employers, a question arises as to how similar is similar enough for relevant comparison.

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[69] 29 C.F.R. § 1607.4D (1984).

Population pool analysis, a variation of applicant flow analysis, compares the number of women or minorities in the employer's work force, or a unit thereof, with the percentage of women or minorities in the relevant geographic area. Another variation compares the percentages of protected class members to nonprotected class members who possess the qualification required by the employer (e.g., educational minima) in a particular geographic area to establish a disparate impact. Yet another variation compares the percentage of minorities in the employer's work force (or unit) who have been promoted with the percentage of nonminorities who have been promoted. Demographic comparisons are especially relevant when an employer's past discrimination or current neutral employment policy (e.g., height and weight minima) may be discouraging minorities or women from applying for a job or promotion, and thus fail to be accurately reflected in an applicant flow analysis.

The problems with these tests are manifold and the plaintiff in a disparate treatment case is often faced with a fight over which test is most appropriate, which geographical area or labor market is most relevant, which unit of the employer's work force should be examined, whether the sample size is statistically significant, and how the protected class should be defined. Resolution of these issues will often require the testimony of statisticians, demographers, and other expert witnesses, and conflicting statistical inferences are possible. Use of an inappropriate test, methodology, or set of statistics may result in a decision being overturned on appeal.[70]

Once the plaintiff has established adverse impact, the employer must show that the employment practice or policy is a business necessity. Proving that an employment practice is substantially job-related in not necessarily simple. Virtually all intelligence, psychological, and physical tests used in the hiring and promotion process must be professionally developed and carefully documented by appropriate validation studies in accordance with professional standards recommended by the EEOC.[71] Although Title VII permits disparate impact pursuant to seniority and merit systems, the use of subjective criteria (e.g., interviews or vague performance evaluations) seldom counterbalances discriminatory impact unless qualifications or performance cannot be evaluated on the basis of objective criteria (e.g., selection of dancers for a show). Educational requirements are almost never sustainable as prerequisites for manual or semiskilled employment, or for admission into training programs.

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[70] *E.g.*, Hazelwood School District v. United States, 433 U.S. 299 (1977). In this case, the trial court compared the percentage of black teachers in the Hazelwood school system with the percentage of black students in the school system and held that there was no evidence of disparate impact. The Federal court of appeals reversed, rejecting the trial court's analysis of the statistical data as resting on an irrelevant comparison and ordering the trial court to compare the percentage of black teachers in the school system with the percentage of black teachers in the local labor market composed of St. Louis City and St. Louis County. The U.S. Supreme Court then reversed the appellate court, holding that the appellate court had not considered the fact that an affirmative action program in St. Louis City may have distorted the comparison.

[71] 29 C.F.R. § 1607.5-14 (1984).

APPENDIX 8A: REPRODUCTIVE HEALTH PROTECTION POLICIES

The following appendix contains sample policies for the protection of employee reproductive health obtained from a range of employers and labor unions. While the material that follows is the actual text of employee protection policies received, many of the facilities surveyed described unwritten policies or procedures for the management of exposure to reproductive health hazards that are not included in this document. OTA has not reviewed company activities to determine whether the policies are in fact complied with or are applied uniformly or in nondiscriminatory fashion. It should also be noted that certain of the companies and facilities contacted by OTA that have written policies did not grant permission for OTA to publish those policies.

Some of the more common features of reproductive health protection programs include:

- **Orientation and information sessions:** These aim to alert employees to potential hazards, including reproductive hazards, to which they may be exposed on the job. Employees are instructed on protective measures (e.g., equipment, hygiene) that can be taken in the workplace.
- **Obtaining information on intentions for reproduction:** Employees may be questioned as to their intentions for reproduction and advised accordingly. (Mandatory exclusion of employees who state an intent to reproduce poses legal issues which are discussed in chapter 8.)
- **Elimination of hazards:** An employer may elim-

inate use of a proven or suspected reproductive health hazard. While this strategy improves safety without necessitating exclusionary practices, technical problems, economic constraints, and/or scientific uncertainty may make it infeasible.

- **Monitoring exposure levels:** Where known or suspected hazards exist, employers may attempt to implement surveillance programs in order to monitor worker exposure levels. Such programs, however, may pose numerous difficulties (e.g., monitoring may be technically infeasible, financially burdensome, or intrusive; scientific uncertainty may remain regarding the degree of hazard and threshold exposure levels of specific agents).
- **Job rotation:** Rotation may be voluntary (e.g., at the request of a male or female employee who intends to have children and is concerned about specific agents in the workplace) or mandatory (e.g., rotation of workers whose exposure levels to a known hazard reach a threshold level). Job rotation is, in most cases, temporary and does not involve a reduction in pay.
- **Exclusion:** An employer may institute a policy that excludes workers who express an intent to reproduce from jobs that pose a threat to worker reproductive health and/or to the health of their offspring. Exclusionary policies that are directed solely at pregnant employees, however, generally do not address the problem of agents that exert their effect on the reproductive capacity of the male or female exposed *before* conception occurs. Moreover, the policies have been criticized as discriminatory because they affect only female employees. (See chapter 8.)
- **Recommended/required notification of pregnancy:** It is the policy of many employers to request that female employees provide notice (e.g., to the Employee Health Service and/or Personnel Office) if they become pregnant. Some employers offer a counseling service to pregnant employees to inform them of potential workplace hazards that may jeopardize the pregnancy and/or health of the developing fetus. Others seek the recommendations of the employee's personal physician regarding appropriate employment activities during pregnancy.
- **Counseling of pregnant employees:** Employers may offer a service wherein female employees who become pregnant are given specific health attention (e.g., by a company physician or health officer). The job site of a pregnant employee may be assessed to identify possible hazards to the employee and/or the developing fetus. Where her job is deemed hazardous, temporary rotation may be considered.

Companies

Shell Oil Co.

Shell has an explicit policy for protection of the embryo/fetus in the workplace. Its purpose is to address and/or manage the risk when existing standards, if any, may not be adequate; when releases may occur, despite controls, that could lead to excessive exposure; or when the employee may not know that she is pregnant. The focus of the policy is to provide as much information as is available on the risk to an embryo/fetus through individual counseling of female employees. In hiring women, there is no distinction made on the basis of age, reproductive, or marital status. A woman is informed of the company's assessment of risk and is also urged to consult her own physician for additional advice if she becomes pregnant or is planning a pregnancy.

First, attempts are made to reduce exposure through the use of engineering or other controls. Jobs in which a fetotoxic or teratogenic agent is present are classified according to the potential for exposure to such agents. For example, a class A job is deemed to present no significant risk. Class B jobs may have levels of exposure which pose a potential threat through the mechanism of fetotoxicity. Class C jobs may have levels of exposure which pose a risk through the mechanism of teratogenicity.

The specific criteria for job categorization are as follows:

- Category A—Job assignments that involve substances that have been suggested to have embryofetotoxicity, but for which the Company believes the pattern of evidence does not indicate that the health of an embryo/fetus would be endangered.
- Category B—Job assignments determined by the Company as posing a potential threat to the embryo/fetus as a result of cumulative exposure or possible exposure above normal operating conditions, but where the Company believes the threat to the embryo/fetus prior to detection of pregnancy is not significant.
- Category C—Job assignments determined by the Company as posing a clearly defined risk to an embryo/fetus because of the possibility of early embryo-fetotoxic and/or teratogenic effects occurring before a pregnancy is detected.

Categorization is to be based on both qualitative and, where possible, quantitative assessment of the likelihood that a given substance could produce adverse effects on the embryo/fetus. This is accomplished through a thorough review of the available scientific literature relative to the substance under consideration. Reported effects, if any, are assessed with due consideration for the levels which produced those effects and the comparable levels of exposure in the workplace.

The effectiveness of engineering (or other) controls is factored into the categorization process when we examine existing air-monitoring data as a part of risk assessment.

The risk to the woman who is unaware of her pregnancy is explained in the definition of Category C above. A job may be categorized as C irrespective of the level of exposure should we identify a possibility of an accidental release, spill, or other event which might result in high levels of exposure for a short period.

Although local union contracts and policies may vary as to eligibility for medical transfer, a woman in any job category may ask to be transferred to another job if she is planning to be pregnant or is pregnant. There is no mandatory rule that a woman inform the Company when planning a pregnancy.

In general, the Company's experience to date in assessing risks has been that controls instituted to protect against carcinogenic risk more than adequately protect against adverse effects on the embryo/fetus.

E.I. du Pont de Nemours & Co.

Du Pont uses a four-step procedure for management of female employees of childbearing capability in order to protect the embryo/fetus:

1. Employees who may be affected shall be informed of the possible consequences of exposure to such substances and appropriate safe handling procedures shall be established and communicated.
2. Engineering controls shall be used to the extent practical to reduce and maintain exposure to embryotoxins to acceptable levels. Such controls shall be augmented by administrative controls as appropriate.
3. Whenever engineering and administrative controls are not practical to keep exposure at or below acceptable levels, personal protective equipment, where appropriate, and training for its proper use shall be provided and required to be used by employees who may be affected by such compounds.
4. Females of childbearing capability shall be excluded from work areas where:
   a. there is potential for exposure to an embryotoxin for which an acceptable exposure level cannot be set, or
   b. whenever engineering and administrative controls augmented as appropriate by personal protective equipment are determined to be inadequate to ensure acceptable levels of exposure.

Du Pont scientists have designated seven substances as requiring special controls because of their potential teratogenic effect:

1. **Lead and related compounds:** Level of 5 ugm/m^3 set which corresponds to about 25 to 30 umg/dl in blood.
2. **Ethylene thiourea (ETU):** Oxidizing agent used in curing rubber. No acceptable exposure level established, found in small quantities because it is a byproduct of some chemical processes in Du Pont plants.
3. **Hexafluoroacetone (HFA):** An additive for polymeric products and a byproduct of such production. Acceptable level set at 0.1 ppm TLV. HFA exhibits a male reproductive impairment effect as well as a teratogenic effect.
4. **Dimethylformamide (DMF):** Solvent, absorbed extremely rapidly through the skin, embryolethal.
5. **Dimethylacetamide (DMAC):** Solvent used in spinning processes; like DMF, rapidly absorbed by the skin, teratogen.

   TLV for both set at 10 ppm. Women of childbearing capacity not excluded if no opportunities for absorption through skin are present or if TLV is not exceeded, and if use of protective equipment protects them from exposure of skin to the liquid.
6. **Formamide:** Embryolethal, similar to DMF, absorbed through the skin. TLV of 10 ppm set, treatment of female employees of childbearing capacity same as that for DMF.
7. **2 Ethoxy ethanol:** TLV set at 10 ppm. Some evidence of both male and female reproductive impairment in experimental animals at 5 ppm. TLV is a compromise.

Additional Sources:

Du Pont newsletter, Medical Division, November 1983, "Issue—Reproductive Hazards." Tom Beauchamp, "Du Pont's Policy of Exclusion From the Workplace," *Ethics in Business and Society*, Beauchamp and Childress (eds.), pp. 24-30.

Exxon Chemical Americas

Policy.—The policy of Exxon Chemical Americas regarding toxic substances is to assure that its operations and products do not create unacceptable risks to the health of employees, customers, carriers, and the public, or to the environment. To this end it will:

A. Adhere to all laws and regulations pertaining to toxic substances control which are applicable to the Company's business. If what is needed to avoid unacceptable risks to health and the environment goes beyond legal requirements, to adopt the practices which the Company judges are necessary, and

B. Take a responsible position of its own where guidelines are needed but where controlling laws and regulations do not exist.

In furtherance of this policy, the Company will:

C. Identify the risks from toxic materials used by the Company or produced in its operations and control them by proper equipment design and operation procedures;

D. Specify precautions required in handling, transporting, using and disposing of products supplied by the Company, in accordance with current knowledge, laws, and regulations;

E. Seek and evaluate new and extended knowledge about the toxic effects of materials manufactured, used and sold by the Company, and share promptly any significant properly evaluated finding with employees, customers, the scientific community, government agencies, and the public; and

F. Work with government agencies and others, as appropriate, in the development and implementation of standards, laws, regulations, and other measures that are needed to achieve satisfactory protection of health and the environment.

The Company's policy is based on the recognition that any substance can be harmful depending on the circumstances of its use or exposure. Since it is not possible to have a "no-risk" environment, the realistic objective is the elimination of unacceptable risks. Historically, society has accepted some level of risk if sufficient safeguards are taken and sufficient benefits are obtained. Ultimately, it remains the responsibility of appropriate public officials to determine what situations are too important to be left to individual choice and in those cases to determine levels of acceptable risk based on competent scientific, economic, and social evaluations.

Guidelines for Implementation of Policy Regarding Toxic Substances.—It is the intent of Exxon Chemical Americas' Policy Regarding Toxic Substances that its facilities will be operated and its products supplied in a manner designed to protect employees and the public from unacceptable risk due to toxic substances. In cases where it is not possible to control such risks by proper designs or practices, the manufacture or use of such materials should cease. Any required precautions associated with the handling of products sold by the Company or its affiliate should by provided by product labels and other means as appropriate. If management has reason to believe that such products are being used in ways that may produce unacceptable risks, it should emphasize to the user the necessity of following the advice for proper practices that has been provided. If subsequent control of the risk is known not to have been achieved, additional appropriate action should be taken.

The primary responsibility for assuring that operations are conducted in accordance with the Company's policy rests with the product lines and operating organization. Managers at all appropriate levels are expected to keep informed on the subject of risks from toxic substances. They are to monitor activities under their supervision, identify and control toxic risks in accordance with the policy, and keep higher management properly informed of any adverse situation regarding materials used or sold by the Company or its affiliates.

Much remains to be learned in defining the parameters of toxicity, and accordingly, managements must be alert to new information and changing circumstances. Sensitivity to the scope and changing nature of toxicity problems and good judgment in seeking solutions to them are required.

Guidelines for Handling Reproductive Risks in the Workplace.—A developing body of scientific evidence indicates that some exposures of humans to such environmental factors as personal lifestyle choices, drugs, certain chemicals, and physical agents such as ionizing radiation can lead to reproductive effects in both males and females. These effects may result in infertility, miscarriages, embryotoxicity, birth defects, and changes in genetic material capable of being inherited. There is particular concern about exposures to the fetus, since it may be especially susceptible to the effects of external agents at exposures which have no effect on an adult. Moreover, an embryo often is most vulnerable to the effects of toxic substances during its earliest development, perhaps even before the mother-to-be is aware of her pregnancy.

Currently, no well-defined or generally accepted approach to the prevention of reproductive risks to employees exists because of scientific uncertainties and differing public opinion. However, the Company has a moral obligation to concern itself with the potential reproductive effects of substances or agents used or produced in its operations.

In accordance with the policy on Toxic and Hazardous Substances and in recognition of the Company's obligation to provide healthful working conditions, the Company's guidelines to reduce the potential for reproductive hazards in their workplaces are outlined below:

A. Review operational and associated biological, chemical, and physical workplace exposures in light of the best presently available information to identify those that might have the potential to be a reproductive hazard.

B. Inform all exposed employees of any potential hazards to the reproductive system from toxic substances to which they are exposed and educate them in the use of personal protective equipment and safe work practices.

C. Control the exposure to such potential hazards to acceptable levels for all employees through the best combination of:
   1. process or equipment engineering designs,
   2. work practice arrangements (such as shortened exposure times where necessary), and
   3. personal protective equipment.

   When there is insufficient basis for the scientific definition of an exposure level with an acceptable reproductive risk, the Medical Department will designate an interim standard which incorporates an appreciable safety factor, and will seek the development of information required for a "permanent" standard.

D. In cases where certain employees are particularly susceptible to the known toxicity of a specific agent, and where exposure cannot be controlled to acceptable levels, implement the indicated protective work assignment practices, including, if necessary, total restriction from potential exposure.

E. Seek on a continuing basis new information on the potential reproductive toxicity associated with manufacturing processes and materials produced, used, transported, and sold by the Corporation.

F. Terminate the manufacture or use of such toxic substances where it is not possible to prevent unacceptable risks to reproductive functions.

Communication Guidelines.—This guideline is intended to further clarify communications requirements of the policy regarding Toxic and Hazardous Substances. Specifically, the following communications requirements relate to information obtained by completion of significant scientific studies of toxic or hazardous substances (such as TSCA 8(e) requirements) or occupational health (e.g., employee epidemiology studies):

1. ECA Management Committee will review plans for and results of studies at critical decision points.
2. ECA will communicate study results and handling recommendation to co-producers and appropriate customers concurrent with release of significant information to appropriate government agencies.
3. Worldwide implications of studies and communication needs will be developed in cooperation with Exxon Chemical's headquarters function and appropriate product lines.
4. Results, including recommended exposure limits, safe handling recommendations, and potential impact will be communicated clearly to all exposed and interested employees.
5. ECA will initiate and/or support publication of completed and internally cleared study results in major scientific journals after peer review.
6. Press release or response statement (with Q's and A's) will be developed and distributed as appropriate.

Related Policies Include "Medical" and "Personnel Safety."

Another Corporation*

This company does not have a fetal protection policy as such. Instead it has implemented procedures for evaluating the risk of exposure to reproductive or developmental (i.e., teratogenic, fetotoxic) health hazards. The following is their description of their objectives and activities:

Reproductive Health Activities.—This company has an established objective of providing a safe working environment for all employees which encompasses the reproductive health of men and women and the fetus. The company has undertaken several steps to achieve this objective:

1. It has developed a computerized data base of citations taken from standardized reference sources where reproductive impairment has been evaluated. These include:

Reference	*Author*
Catalog of Teratogenic Agents	Thomas H. Shepard, M.D.
Reproductive Hazards of Industrial Chemicals	S.M.Barlow/F.M. Sullivan
Chemical Hazards to Human Reproduction	Ian C.T. Nisbet/ Nathan J. Karch
Handbook of Teratology	James G. Wilson/ F. Clarke Fraser
Occupational Chemicals Tested for Teratogenicity (Int. Arch. Occup. Environ. Health)	K. Hemminki
Health Effects of Environmental Chemicals on the Adult Human Reproductive System (Toxicology Information Response Center)	J.G. Pruett/S.G. Winslow
Registry of Toxic Effects of Chemical Substances (Reproductive Subfiles)	

2. An inventory of all chemicals used or manufactured at each facility has been developed. These

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*This institution/company has asked to remain anonymous.

inventories are then compared with the chemicals listed in the data base.
3. Each chemical that comes up from the cross tabulation is carefully reviewed and analyzed by an experienced toxicologist and a physician.
4. Exposure data are considered, should the literature review indicate a potential hazard to reproductive health.
5. If work practices and engineering controls are insufficient to protect the workers from risk to reproductive health, alterations in work procedure will be implemented. To date, there has been no need for risk management strategies because no chemicals in use or manufacture have been found that pose a sufficient reproductive health risk.
6. For chemicals of significant use at the company for which adequate reproductive toxicology data are unavailable, the company has a toxicology research effort to develop and validate screening methods. The company is doing some of this research in-house and working with various trade associations that are examining the validity of standardized tests for reproductive impairment.

Hospitals

Memorial Sloan-Kettering Cancer Center: Personnel Department Guidelines for Pregnant Employees

Policy.—Pregnancy will be treated as any other illness requiring temporary disability. The policy on temporary disability due to pregnancy is maintained in the personnel Department. Employees receiving temporary disability are paid their full salary, not to exceed 26 weeks.

Purpose:

1. To protect the health of the pregnant employee and her fetus by developing recommendations for her safe placement in a particular job, for her continuing to work as pregnancy develops, and for her return to work following delivery.
2. To promote early recognition of pregnancy as a means of health and safety protection for the pregnant employee and the fetus.

Procedure:

1. After an employee is hired, the Personnel Department orients the new employee to medical insurance and disability benefits.
2. During the Employee Health Service (EHS) preplacement health evaluation and orientation, the employee is informed of the EHS services that are available, including Free Pregnancy Testing.
3. If the Personnel Department first becomes aware of an employee's pregnancy, they:
   a. review the temporary disability policy with the employee, and
   b. refer the employee to the Employee Health Service.
4. If the Employee Health Service Department first becomes aware of an employee's pregnancy, we:
   a. refer the employee to Personnel for the review of policy;
   b. ask for permission to contact the Laboratory Safety Department;
   c. request that the employee and her personal physician complete a Disability Form; and
   d. offer counseling.
5. Representatives of the Laboratory Safety Department inspect the employee's work site and discuss with the employee her daily work activities. Recommendations are then made for possible modifications in work safety practice, transfer, or temporary discontinuance of work. If the two latter recommendations are made, the Personnel Department is notified.

   Recommendations by the Lab Safety Department are made in the interests of the pregnant employee in a way that will help her understand, accept, and use them. If, however, the employee refuses to accept these recommendations, the Lab Safety Department requests her to sign a form indicating that she has been make aware of the potential hazards.
6. When the Disability Form is returned to the EHS:
   a. recommendations by the personal physician are granted if deemed reasonable according to accepted medical practice. If a recommendation does not seem to be reasonable, the EHS may request that the employee obtain a second opinion from a doctor selected by the EHS, at no cost to the employee.
   b. The EHS sends the original Disability Form to the Personnel Benefits Department, Disability Section, and retains a copy for the employee's medical folder.
   c. The nurse makes a notation on the calendar of the employee's first date of inability to work and later transfers it to the Disability List.
7. The EHS requests notification of the date of delivery and sends another Disability Form to the employee which is to be filled out by her and her obstetrician at the 6-week postpartum appointment.

Another Hospital*

Statement of Purpose.—In a complex medical environment employees may work with substances known or suspected of being capable of posing a hazard to human reproduction. The purpose of this policy is to ensure that exposures to these substances pose no significant risk to employees.

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*This institution/company has asked to remain anonymous.

Policy and Guidelines.—This policy is important to employees who are working with substances that may be capable of posing a hazard to human reproduction. A list of substances applicable to this policy is included. Periodic updates of this list will be made by the Environmental Safety Committee.

Exposure levels shall be limited to one-half (½) of the safe exposure guidelines established by the American Conference of Governmental Industrial Hygienists. If no such guideline exists, exposures shall, through engineering and/or work practice controls, be controlled to acceptably low levels of risk for adverse reproductive health effects. When, in the opinion of the Division of Preventative Medicine, the risk of exposure to a substance is such that no exposure can be considered safe, placement shall be determined on a case-by-case basis.

When exposures to the listed substances have been controlled as prescribed in the above paragraphs, employees will be expected to continue in their current job assignment.

Supervisors of personnel having exposures to listed substances should review this policy with their employees and remind them of the need to inform their supervisor in the event they become pregnant. Further information on risks and precautions should be referred to the Division of Preventative Medicine, or the Personnel Section's Environmental Safety Coordinator.

Reproductive health risks associated with infectious agents are addressed by the Infection Committee in institutional isolation procedures. Questions should be referred to the Chairman of the Infection Committee.

Reproductive health risks associated with ionizing radiation are addressed by the Radiation Safety Office in accordance with guidelines established by the U.S. Nuclear Regulatory Commission. Questions should be referred to the Radiation Safety Officer.

Substances:

Adriamycin
5-Azacytidine
Benzene
Cadmium
Capafol (Difolatan^{R})
Captan
Carbaryl
Diethylstilbestrol
Dibutylphthalate
Dimethyl acetamide
Dimethylphthalate
Dioxin
Diphenylamine
Diquat
Ergotamines
Carbon monoxide
Carbon tetrachloride
Chlorambucil
Cyclophosphamide
Dacarbazine
Diazinon
2,4-dichlorophenoxyacetic acid (2,4-D)
Mercury compounds
Methoxyfluorane
Methyl cellosolve
Methotrexate
Nitrous oxide
Paraquat
Parathion
Procarbazine
Ethylene dibromide
Ethylene oxide
Ethylene thiourea
Hexafluoroacetone
Indium and compounds
Lead compounds
Melphalan
Thiotepa
Thiram
2, 4, 5-Tricholorphenoxyacetic
(Agent orange)
Vinblastine
Vinyl chloride
Warfarin

Walter Reed Army Medical Center: Medical Service Occupational Health Program

Purpose.—This regulation outlines policies and procedures for the implementation of the occupational health portion of the WRAMC Occupational Safety and Health Program.

Scope:

A. Program elements are listed in appendix 8A-1.

B. The provisions of this regulation apply to assigned and attached elements of WRAMC, its tenant activities, and to other commands, installations, and activities provided occupational health support by WRAMC. This regulation should be incorporated by reference into applicable local regulations. Support Agreements, Memorandums of Understanding, and similar agreements supersede the provisions of this regulation while they are in force. The term "employee" refers to both military and civilian personnel, unless stated otherwise.

C. Specific applicable procedures for the Hearing Conservation Program (WR 40-62) and the Occupational Vision Program (WR 40-14) will be found in the indicated regulations.

General:

A. Commanders are responsible for the establishment, implementation, and overall supervision of the occupational health program at supported facilities.

B. Items of protective clothing and equipment required to comply with safety and occupational health regulations and procedures shall be furnished to military and civilian personnel at no cost to these personnel.

C. A desire and a willingness to utilize protective clothing and equipment should be stimulated among personnel by an educational program to include formal discussion, films, and the use of posters. Safety awards may increase motivation. Habitual nonuse of protective clothing and equipment, engineering controls, and violation of SOPs should be considered grounds for disciplinary action.

D. WRAMC occupational health personnel will participate in health maintenance and health promotion activities to the maximum extent possible;

however, whenever employees are exposed to occupational health hazards, priority for available resources must be given to prevention, detection, and correction of occupational health illness and injury, as required by law and by regulation.

Responsibilities:

A. Commanders at every echelon shall ensure that:
  1. The working conditions for each employee, civilian and military, have been evaluated for occupational health hazards.
  2. Appropriate engineering controls and/or protective clothing and equipment are provided.
  3. Each employee, civilian and military, is enrolled in an appropriate medical surveillance program.
  4. Periodic inspections are conducted to ensure compliance. Appropriate corrective measures are instituted.
  5. Each employee is given information regarding health hazards associated with his job, relevant medical symptoms, appropriate emergency treatment, and the employee's responsibility for using protective clothing and equipment.

B. Preventive Medicine Activity, WRAMC will:
  1. Provide occupational medicine consultation.
  2. Complete and periodically update an Inventory of Occupational Health Hazards.
  3. Conduct industrial hygiene surveys to evaluate operations or practices involving actual or potential occupational health hazards. Assign and report Risk Assessment Codes for health hazards to the appropriate Safety Officer.
  4. Conduct epidemiologic investigations when situations develop suggesting the possibility of an increased disease or injury rate attributable to occupational hazards.
  5. Assist commanders in providing employee health education by provision of lesson plans, lecturers, and loan of health education materials.
  6. Provide physician review of medical monitoring recommendations for employees serviced by the WRAMC Occupational Health Clinic and the Civilian Employees Health Service, DOD (CEHS).
  7. In conjunction with Chief, WRAMC Department of Primary Care and Community Medicine (DPCCM) will:
    a. Conduct job-related health examinations including preplacement, periodic, and administrative examinations. Voluntary health maintenance examinations, such as screening for high blood pressure, diabetes, glaucoma, etc., will be conducted as personnel and other resources permit.
    b. Provide limited treatment of illness and injury.
    c. Conduct illness absence monitoring:
      i. Employees should be required to clear through the servicing occupational health clinic facility prior to departure from work because of illness to insure they receive adequate medical care, to permit detection of illness caused by work conditions, and to conserve lost man-hours where palliative treatment will permit the employees to remain on the job.
      ii. Employees also should be cleared through the clinical facility prior to returning to work after an illness in excess of 5 working days to ensure they are not returning to work before being physically able, will not be adversely affected by exposures to health hazards (e.g., unable to wear a respirator), or pose a risk to other employees with chronic diseases or disabilities who may affect or be affected by their work assignment.
    d. Conduct Chronic Disease or Disability Surveillance. Identify and maintain a list of employees with chronic diseases or disabilities who may affect or be affected by their work assignment.
    e. Conduct an Immunization Program. Appropriate immunizations will be provided employees potentially exposed to infectious disease because of the work environment or required foreign travel. Influenza vaccine immunizations will be made available annually. Guidelines for administration of specific immunizations are given in HSC Pam 40-2.
    f. Conduct a Pregnancy Surveillance Program. Pregnant workers, military and civilian, are encouraged to report to the clinical facility as soon as pregnancy is determined so that the impact of work conditions upon the pregnancy can be evaluated, and protective measures prescribed. This surveillance will not supplant care provided by the employee's personal physician.
    g. Conduct an Alcohol and Drug Abuse Prevention and Control Program. Evaluation, diagnosis, counseling, and referral will be conducted in conjunction with established command, installation, and activity programs.
    h. Provide Employee Health Education. One-to-one health counseling on both job-related topics and general health maintenance will be conducted during nursing appraisals and

health examinations. Group general health maintenance and health promotion activities will be provided upon request to the servicing occupational health facility and as resources permit.

  i. Prepare and maintain appropriate medical records, and Army and Occupational Safety and Health Reports.
  j. Maintain master schedules by work location for, and schedule, medical surveillance.

C. Chief, WRAMC Department of Primary Care and Community Medicine (DPCCM), will:
  1. Discharge those joint responsibilities indicated in subparagraph 4b(7).
  2. Provide physician review of medical surveillance recommendations for employees serviced by the WRAMC U.S. Army Health Clinics.

D. Civilian Personnel Officers will:
  1. Provide periodic updates to servicing occupational health clinical facilities regarding terminations, new hires, and transfers.
  2. Maintain the following inventories:
    a. Job categories requiring specific levels of physical fitness for the employee to perform effectively and with safety to himself/herself and others, e.g., firemen and mobile equipment operators.
    b. Job categories which involve exposures to occupational health hazards.
  3. Ensure personnel applying for positions in job categories requiring a minimum level of physical fitness are referred to the supporting occupational health clinical facility for preplacement examinations.
  4. Ensure that each new employee assigned to positions involving occupational health hazard exposures processes through the supporting occupational health clinical facility so that appropriate medical baseline examinations can be conducted and a medical record initiated.
  5. Incorporate physical fitness requirements, and requirements for utilization of personal protective equipment into job descriptions, as appropriate.

E. Safety Officers will:
  1. Assume responsibility for overall conduct of the OSHA Program in their area of responsibility, as delegated by Commanders.
  2. Implement safety aspects of the organization's OSHA program to include:
    a. Validation of requests for protective clothing and equipment.
    b. Inspection of workplace environments utilizing Standard Army Safety and Occupational Health Inspection (SASOHI) procedures, if applicable.
    c. Management of the Army Hazard Reporting System, when applicable.
    d. Preparation and monitoring of the Installation Hazard Abatement Plan (DA Form 4756, for Army installations) or variance for each hazard identified with Risk Assessment Code (RAC) of IIIB or higher, not correctable within 30 days.
    e. Conduct of job safety and health training.
  3. Complete all OSHA-required reports except as noted in para 4b(7) and 5d.

F. Supervisors will:
  1. Schedule employees for medical examination when appropriate (such as, when notified periodic medical examinations are due, for new employees, when employees return from sick leave in excess of five (5) days, and when fitness for duty examinations are required).
  2. Ensure personal protective equipment is utilized when necessary, and that action is initiated to evaluate and/or abate a hazard occurring in the workplace.
  3. Initiate adverse personnel actions when necessary to ensure compliance with applicable Occupational Safety and Health rules and regulations.

G. Employees will:
  1. Comply with requirement established under the provisions of OSHA to assure a safe and healthful working environment.
  2. Utilize protective clothing and equipment provided, and report for scheduled medical examinations and health and safety training.
  3. Report unsafe and unhealthful working conditions.

Procedures:

A. Inventory of Occupational Health Hazards:
  1. The inventory will include, as a minimum, information required by the Occupational Safety and Health act (OSHA):
    a. Location.
    b. Description of the operation and the number of employees involved.
    c. Exposure information, both actual and potential, to occupational health hazards including type and degree of exposure, and documentation of exposures approaching or exceeding national consensus standards for a hazard.
    d. Description of controls utilized to reduce or eliminate employee exposure.

    e. Identification, by name and SSN, of employees exposed at each location.
2. The inventory will be completed and updated in accordance with an Industrial Hygiene Implementation Plan (IHIP) prepared annually to satisfy WRAMC Occupational Health Program goals.
3. Access to information in the inventory will be restricted under the provisions of the Privacy Act as specified in AR 340-18-9. Copies will be provided the servicing occupational health clinical facility with extracts provided Safety, Civilian Personnel Officers, and others upon request.

B. Health Examinations:

1. In the absence of completed occupational health hazard inventories, physicians authorized to establish medical surveillance requirements should utilize work location, work history, and the following references to specific requirements:
    a. Appendices E, G, and H, Medical Surveillance Guide, USAEHA.
    b. DOD manual 6055.M, Occupational Health Surveillance Manual.
    c. TB Med 279, Control of Hazards to Health from Laser Radiation.
    d. TB Med 501, Hearing Conservation.
    e. TB Med 502, Respiratory Protection Program.
    f. TB Med 506, Occupational Vision.
    g. TB Med 523, Control of Hazards to Health from Microwave and Radio Frequency Radiation and Ultrasound.
2. Upon completion of the inventory, physicians will be provided recommendations for medical surveillance by the WRAMC Preventive Medicine Activity, tailored to significant exposures in an employee's job. Physicians are encouraged to minimize laboratory support requirements and health examination complexity so that utilization of occupational health nurse expertise can be maximized and employee lost time minimized. Guidelines for minimum physical examination requirements are given in HSC Pam 40-2.
3. The only personnel authorized to establish, or modify, medical surveillance protocols are: Deputy for Preventive Medicine Activities; Chief, Department of Primary Care and Community Medicine, for Army Health Clinics; and Director, Civilian Employees Health Service, DOD. Medical personnel other than those physicians specifically designated as responsible for establishing medical surveillance requirements are not authorized to make revisions to an individual's health examination protocol without specific written permission. Apparent discrepancies between work history and the health examination protocol will be referred to the WRAMC Preventive Medicine Activity for resolution. Discrepancies will not serve as an excuse to delay implementation of the established protocol.
4. New employee and periodic health examinations will be performed at the servicing occupational health clinical facility by assigned, and qualified, occupational health nurses to the greatest extent possible. These examinations will be given priority over walk-in visits for nonoccupational illness and injury. Employees will normally be referred for physician examination when special, preplacement, requirements exist, and when toxic chemical exposures are involved and will be referred to servicing medical laboratories for laboratory work. Alternative arrangements for the purpose of reducing employee lost-time for laboratory visits, such as utilization of local Agency resources for collection and delivery of laboratory samples, are encouraged.

C. Treatment of Illness and Injury:

1. Civilian employees on TDY status are eligible for treatment.
2. Employees with job-related illness and injury will be provided or compensated for (under Federal Employee Compensation Act rules and procedures, or equivalent programs for military personnel) emergency and follow-up care.
3. Emergency treatment and limited palliative treatment of both occupational and nonoccupational conditions is provided to prevent loss of life, relieve suffering, or reduce absenteeism, with referral to personal physician or other health resources as appropriate. The capability to provide treatment for illness and injury is extremely limited. Neither staffing nor equipment are available to provide full shift coverage or more than basic CPR emergency support.
4. Unless located at installations having after-hours emergency health care facilities, however, care should be sought from the servicing Fire and Rescue Service, or the nearest civilian emergency treatment facility.
5. First aid kits are not normally considered acceptable and will be procured and equipped only with the authorization of the Deputy for

Preventive Medicine Activities, WRAMC. Conditions under which such kits may be authorized include industrial locations where either fast-acting, highly toxic chemicals are in use which require specific treatments and antidotes to be readily available, or significant waits could be expected before the arrival of ambulances during hours when the servicing occupational health clinical facility is closed. In each case, kits must be assigned to individuals currently certified as having acceptable first aid training (e.g., American Red Cross Courses).

D. Medical Records:

1. A civilian employee medical record will be initiated and maintained on all civilian employees identified by CPO as belonging to a job category or by LOHHI as involving occupational exposure, including permanent Nonappropriated Fund employees. Utilization of DA Form 3444 (Terminal Digit File for Medical Record) is not authorized. Civilian employee medical records will be maintained separately from military medical records, and will normally be maintained in the occupational health clinical facility directly servicing the employee's work area.
2. Medical records of Active Duty (AD) military personnel will not be maintained in the servicing occupational health clinical facility. The medical record will be flagged with a small sticker to indicate that the individual is occupationally exposed to significant health hazards. The stickers, and explanatory fact sheets requesting reporting of job-related illness and injury to the occupational health facility, will be provided by the WRAMC Deputy for Preventive Medicine Activities. Clinical facilities are encouraged to initiate and maintain a record on military personnel containing, as a minimum, HSC Form 79 (Master Problem List), and DD Form 2005 (Privacy Act Statement-Medical Records).
3. Medical records of dual status personnel will be handled the same as military medical records, when possible, to include flagging. If the individual refuses to bring the military medical record to the occupational health clinical facility, medical records may be maintained until such time as the medical record becomes available. The individual should be provided a copy of SF 600 (Chronological Record of Medical Care) for placement in the military medical record. A distinctive mark, such as a "D," may be used as a flag.
4. An additional distinctive mark, such as white tape, may be used to indicate records of personnel with chronic disease and injury problems.

E. Army and Occupational Safety and Health Records and Reports:

1. The Army Occupational Health Report (DA Form 3076) will be prepared by each clinical facility providing occupational health services and submitted NLT the 3rd working day of the month following the end of a semiannual reporting period to the WRAMC Preventive Medicine Activity (ATTN: HSHL-HO). Daily occupational health workload data will be collected utilizing DA Form 3075 (Occupational Health Daily Log), or its equivalent.
2. OSHA Form 100F (Log of Federal Occupational Injuries and Illnesses) will be maintained by each clinical facility providing occupational health services and submitted as requested by the servicing CPO or Safety Office.
3. Other records will be maintained as necessary for time accounting, billing, and other purposes as specified in applicable Standing Operating Procedures. Duplication of recordkeeping efforts will be avoided.

F. Medical Surveillance Scheduling:

1. Master schedules will be prepared by the servicing occupational health clinical facility for medical surveillance scheduling. Schedules should be based on Local Occupational Health Hazard Inventories (LOHHI) provided by Deputy for Preventive Medicine Activities, and should be organized so that an entire department, section, or organization is scheduled within a short time period.
2. The clinical facility will notify supervisors, in writing, when medical surveillance examinations are required. The attached form (appendix 8A-2) may be utilized, and need not be typewritten. A log of notifications should be maintained so that second notices may be sent if scheduled personnel fail to keep their appointments.
3. The clinical facility will notify its next higher organizational element (DPCCM, Deputy for Preventive Medicine Activities, CEHS) of second failures to keep appointments. This element should then notify, in writing, applicable Headquarters elements of the failure so that appropriate administrative measures may be taken.

References

A. WR 40-14, Occupational Vision.
B. WR 40-62, Hearing Conservation.
C. HSC PM 40-2, Occupational Health Program.
D. AR 40-5, Health and Environment.
E. AR 385-32, Protective Clothing and Equipment.

Appendix A-1: Occupational Health Program Elements (by priority)

1. Required by law and regulation.
    a. Inventory of Occupational Health Hazards and Listing of Positions Requiring Special Physical Fitness Standards.
    b. Job-related Medical Surveillance-Preplacement/Reassignment, Periodic, Termination, including vision and hearing conservation screening.
    c. Treatment of Occupational Illness and Injury.
    d. Employee Education Regarding Job Hazards.
    e. Safety and Health Inspections.
    f. Medical Records.
    g. OSHA Record/Reports.
    h. Medical Directives.
    i. Alcohol and Drug Abuse Prevention and Control.
2. Required by Regulation.
    a. Industrial Hygiene Survey.
    b. Administrative Examination-Fitness for Duty, Return After Illness, Disability Retirement.
    c. Elective Periodic Vision Screening.
    d. Emergency/Palliative Treatment of Nonoccupational Injury.
    e. Sickness Absence Prevention.
    f. Chronic Disease Surveillance.
    g. Pregnancy Surveillance.
    h. Job-Related Immunizations.
    i. Epidemiologic Investigations.
    j. Occupational Health Reports, Local Regulations and Supplements, and Standing Operating Procedures.
3. Elective:
    a. Voluntary Health Maintenance Evaluations-Medical Examinations, Nursing Health Appraisals, Specific Disease Screening.
    b. Non-Job-Related Immunizations.

Appendix A-2

Your organization is scheduled to report for medical surveillance examinations during the month of __________________. Request you contact this occupational health clinical facility at ________________ to schedule the individuals named below for medical surveillance.

Occupational Health Nurse

Labor Unions

United Steelworkers of America

The following policy of the steelworkers combines the preventive aspects of industrial hygiene, medicine, and law in a manner designed to maximize the occupational health and equal employment opportunities for all workers, including those capable of having children:

"Policy on Potential Reproductive Hazards"

A. It is the goal of the Company to fully protect the reproductive health of male and female employees, and to eliminate any risk of damage to unborn children. The Company recognizes that there are several steps that may be taken when exposure to a toxic substance poses a risk to the reproductive health of employees, or to their unborn children. The best alternatives are the replacement of the substance by a safer material; the installation of effective engineering controls, such as enclosure and local exhaust ventilation; and the use of safer work practices. While the transfer of certain male or female employees may be necessary in some cases, it will only be considered where:
    1. Substitution, additional engineering controls, and safer work practices are technologically infeasible or ineffective in reducing exposure to the desired levels, and;
    2. The risk of reproductive damage is confined to the group to be transferred.

B. Wherever the Company has reason to believe that a particular substance or substances may pose a risk to the reproductive health of male or female employees, or to their unborn children, the Company will inform the Union and will, prior to any action, discuss with the Union the reasons for its beliefs (with documentation, if requested) and the steps to be taken.

C. When a determination is made that exposure to a particular substance poses a risk to the reproductive health of male or female employees, or to their unborn children, the Company will replace the substance with a safer material, or will install all feasible engineering controls, and institute safer work practices, in order to reduce exposure to safe or lowest feasible levels. Such steps will be taken even if certain employees are also transferred from the particular job or department.

D. If it is decided that certain employees must be removed from exposure, then the group of employees affected will be defined as narrowly as possible, taking into account the risks of the particular substance, while providing for the greatest possible element of employee choice consistent with adequate protection of reproductive health and the health of unborn children.

E. No employee removed as a result of this policy will suffer any loss of earnings. Transfers will take place according to existing seniority arrangements. Transferred employees will receive the earnings applicable to the new job, or to the former job, whichever is higher.

F. The Company will provide proper medical surveillance to employees exposed to occupational hazards.

G. The Company will maintain an adequate research program, in order to determine the reproductive and other effects of the substances to which employees are exposed.

H. The Company will not discriminate by sex, race, or age in the hiring or promotion of employees because of alleged differences in susceptibility to reproductive effects caused by toxic substances.

International Chemical Workers Union: Policy on Reproductive Effects of Hazardous Materials

Introduction.—During 1977, several companies announced policies that would remove women of childbearing age from certain departments or jobs. Such policies aim to limit exposure to ". . . chemical agents which may have the capacity to cause developmental defects in unborn fetuses." (The scientific term for such a chemical which affects an unborn fetus is "teratogen.") These same policies would also prevent women from bidding on future openings for jobs in those departments.

In dealing with reproductive hazards, labor unions are faced with three major concerns. First, many teratogens are also "mutagens"—agents that can alter the genetic make-up of the chromosomes contained in the human egg and sperm. This means that future generations might carry new or "mutant" characteristics which could be detrimental but may remain hidden for some generations. Damaged chromosomes from either parent could also cause birth defects or spontaneous abortions. In addition to genetic damage, reproduction functions may also be affected. Sterility may occur or there may be an inability of the sperm and egg to conceive a new individual.

Secondly, teratogens and mutagens may also be "carcinogens," chemicals that are known to cause cancer. It is therefore essential that chemicals that pose a reproductive hazard be controlled as if they were suspected carcinogens.

A third major concern for labor unions is the emphasis that companies have placed on protecting a developing fetus. This concern is based on the companies' fear of third-party liability. An injured child might well file suits against a company for damages resulting from the mother's occupational exposure during pregnancy. Rather than risk such third-party liability, companies are choosing to bar and remove women of childbearing age from exposure to chemical hazards regardless of the scientific basis for such actions.

Company policies, however, do not address the fact that birth defects from chromosomal damage can be passed along after women are removed from hazardous exposure. Also, despite the fact that chemical mutagens can attack the genetic materials of men and women equally, companies have addressed reproductive hazards as if they only affected women. Some companies are trying to deliberately drive a wedge between men and women workers with the ultimate objective of eliminating women from the workplace. Companies take advantage of normal male feelings which tend to protect women and mothers, and, on the other hand, normal female emotions which may lead women to relinquish their jobs and job rights in order to protect their unborn children.

Companies, however, assume additional liabilities under EEOC if women are discriminated against because they have unjustly been denied equal employment opportunities, promotions, and even jobs. *There is also a potential discrimination claim by the men who continue to be exposed after the women are removed.* A union may also be liable if it does not successfully provide for a safe working environment through collective bargaining and administration of the agreement; that is, a liability for failure to fairly represent employees. In addition to the reproductive hazards from exposure to teratogens or mutagens, there may be other harmful health effects. *The union cannot ignore its responsibilities to bargain for a safe and healthful working environment for all its members, regardless of sex.* Allowing women to be arbitrarily barred from a workplace because of a reproductive hazard is an inadequate solution in protecting the health of *all* workers. Our policy therefore must be broad enough to protect all of our members, while allowing for the resolution of specific problems. The following policy should provide general guidance to our field staff and local union officers who will be first confronted with company policies or scientific evidence regarding reproductive hazards.

Policy.—The International Union will require its subsidiary bodies to follow the following procedures when they are faced with the announcement by an employer that females will no longer be allowed to apply for or retain a specific job or work in a specific department or on a special process:

1. **When an ICWU local union receives notice from the employer about a change involving sex-related hazardous exposures, the Regional Director, the ICWU Health and Safety or Legal Department should be contacted immediately and before an official reply is given to the company.**

2. Usually the union is informed orally about the employer's decision. A written request should immediately be made to the employer asking them to meet officially with the Union Committee to bargain over the appropriateness of the company's decision and the effects of that decision. The request should also ask for written justification of the employer's position and all information pertinent to the decision (air-monitoring data, scientific literature, results of medical surveillance of all exposed employees, etc.).
3. a. Regardless of whether or not the data are inconclusive or inconsistent with the employer's position, we should demand that the employer bargain on the issue; or
    b. A grievance should be filed on the matter without undue delay so that our rights to contest the proposed change will be protected.

NOTE: Any refusal by the employer to meet with the union, to provide requested information, bargain on the issue, or process a grievance should be communicated immediately to the International President and the ICWU Legal Department.

4. If the employer's announcement comes during negotiation of a new agreement or at a time just prior to negotiations, we must deal with the issue in the negotiations. **Again, the ICWU Health and Safety and/or Legal Department must be advised. Contract proposals and advice will be provided.**
5. Before any final action is taken, we may seek plant inspections by the Occupational Safety and Health Administration (OSHA) and/or a Health Hazard Evaluation performed by the National Institute for Occupational Safety and Health (NIOSH) in an effort to secure the best possible data for our final position regarding health and safety matters.
6. The ICWU Collective Bargaining Department will provide collective bargaining advice and agreement language for negotiations which will be specifically designed to protect the rights of our local unions and members and ensure relief from the undue hazardous exposures that are specific to the particular local union. General agreement language can be found in the *ICWU Health and Safety Guide for Local Unions*.

It is the position of the International Chemical Workers Union that worker exposure to hazardous materials should be reduced to zero or at least to the lowest technologically feasible level. Separate exposure levels for men and women would not provide a safe and healthful workplace for all workers.

In most cases, engineering controls and process technology are available to industry which will reduce, if not totally eliminate, hazardous exposures. Unfortunately, industry usually responds with inflated cost estimates and proposals that workers be encapsulated in respirators or full-body protective devices. The OSH Act of 1970 recognizes the use of personal protective devices as only a temporary solution. The implementation of engineering controls is the only acceptable final solution for the control of hazardous materials.

We believe it is within the capacity of industry to provide a workplace free of recognized hazards for both men and women. This union, therefore, rejects and challenges any company policies which would remove or bar women from any employment opportunities available to men in plants under contract to ICWU.

Chapter 9

Workers' Compensation

CONTENTS

Page
Introduction 279

Workers' Compensation 279
  Occupational Disease Compensation 280
  Classification of Workplace Reproductive Injury and Disease 280
  Criteria for Securing Benefits for Reproductive Harms 288
  Conclusion 296

List of Tables

Table No. *Page*
9-1. Summary of Occupational Disease Coverage by State 281
9-2. Eligibility for Compensation for Reproductive Harms: Personal Injury Criterion 289
9-3. Summary of Harms, Victims, Benefits, Criteria, and Causation Problems . . . 293

Chapter 9

Workers' Compensation

INTRODUCTION[1]

Health hazards in the workplace are governed by various sectors of law. Regulatory agencies are authorized to *prevent* hazards, whereas workers' compensation statutes and the tort litigation process are used primarily to *provide compensation* for injuries and diseases. Workers' compensation laws (and, to a lesser extent, tort law) are also intended to deter hazardous conduct by threatening employers with liability to injured workers or increased insurance costs. The failure of workers' compensation laws to have a significant deterrent effect resulted in the creation of the Occupational Safety and Health Administration (OSHA) and other agencies with the authority to mandate safe workplace conditions.

This chapter discusses State workers' compensation systems as a vehicle for compensating workers who have been reproductively harmed in the workplace; chapter 10 discusses the tort liability system. **Both the workers' compensation and tort liability systems fail to consistently provide compensation to the victims of occupationally induced reproductive failure, though they sometimes result in some compensation for some workers.**

WORKERS' COMPENSATION[2]

Each of the 50 States requires that employees of private firms be covered by a form of insurance known as *workers' compensation*. Under the workers' compensation systems now in effect, workers are entitled to receive monetary compensation from their employers for wage losses, medical expenses, and other costs incurred as a result of injury, disease, or death arising from certain job-related circumstances. Although workers' compensation is the sole official source of compensation for injured or diseased workers, it is frequently criticized as providing inadequate benefits to employees and insufficient incentive for employers to offer more healthful workplaces.

Unlike a claimant in the tort system,[3] the worker who seeks workers' compensation need not prove employer fault or negligence, nor establish that the worker was without fault or negligence. The defendant (employer or insurer) may accept the claim, try to settle, or contest the claim. A defendant typically contests a worker's compensation claim by contending that the injury did not arise while the worker was acting within the scope of employment, that the worker's injury is not covered by the compensation statute, or that the worker was not injured at all. If a claim is contested, an evidentiary hearing is held before a State board. In most States, the board decision may be appealed to a special appeals board and/or to a court by either party.

Monetary benefits may be *scheduled* or determined by a *formula*. Scheduled benefits provide a specific sum usually determined by statute for the specific injury. Formula benefits provide workers who have permanent or temporary, total or partial disabilities with income maintenance at a level determined by the statutory formula during the period of disability.

Although workers' compensation laws vary from State to State, certain attributes are common among State statutory schemes. The foremost attribute, criticized by those who consider the system inadequate, is the *exclusivity of remedy* doctrine, discussed later, which provides that an employee covered by a workers' compensation statute cannot sue his or her employer at common law for any injury or disease subject to the statute. (Employee suits against the manufacturers of dangerous products used by their employers are not affected by the exclusivity doctrine. Also unaffected by the exclusivity rule are suits by the employee's spouse or offspring, since these nonemployees are not covered by workers' compensation laws in the first place.) This barrier to tort actions by diseased or injured workers against their employers has been eroded in several States, which now permit tort actions against employers in limited circumstances. **Nevertheless, workers' compensation is the sole official source of compensation for most injured or diseased workers.**

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[1] This chapter relies in part on a contract report prepared by Michael Baram, Professor of Health Law, Boston University. M. Baram, Reproductive Hazards in the Workplace: Workers' Compensation Law (May 1984) (unpublished report).

[2] See generally P. Barth, Workers' Compensation and Work-Related Illnesses and Diseases (1980); A. Larson, The Law of Workmen's Compensation (1978); Workmen's Comp. L. Rep. (CCH).

[3] A *tort* is a private or civil wrong or injury, other than breach of contract, for which a court will permit recovery of monetary damages and/or other forms of relief.

Occupational Disease Compensation

Workers' compensation laws were initially enacted to deal with the "easy case": compensating employees with injuries caused in accidents. Later, recognition of occupational diseases and the filing of disease claims led to expansions of coverage. Today, the workers' compensation law in every State is applicable to occupational diseases.[4] Table 9-1 provides a summary of occupational disease coverage provisions.

Only 5 to 8 percent of all workers' compensation claims are claims for occupational diseases, however.[5] Explanations for the small number of disease claims include:

- Workers, medical experts, and attorneys do not readily recognize the job-relatedness and compensability potential of many diseases.
- Some claims may be discouraged because of the difficulty of proving a causal link between a workplace exposure and the disease.
- Some States require disease compensation claims to be filed within a specified period of time (generally, 1 to 3 years) after the most recent occupational exposure, thereby precluding claims for diseases that have long latency periods, such as cancer and some developmental effects.

Classification of Workplace Reproductive Injury and Disease

The reproductive health of the male and female worker, the health of the embryo or fetus carried by the pregnant worker, and the health of the worker's spouse or offspring can be injured or impaired in many ways by occupational circumstances. The workers' compensation system, however, is structured so as to afford coverage and the opportunity for compensation for only some of these harms.

The occupational circumstances leading to possible reproductive injuries and diseases include:

- accidental injuries suffered by the worker or the embryo/fetus (e.g., testicular injury from physical impact, embryo/fetal injury from worker fall);
- physical stress of the worker (e.g., miscarriage arising from heavy physical exertion);
- acute or chronic exposure of worker or fetus to chemical, physical, or biological agents in the workplace that directly result in reproductive damage or loss of sexual capacity; and
- other acute or chronic exposures of workers that lead to primarily nonreproductive injuries or diseases but which, as a side effect, also impair the worker's reproductive or sexual function (e.g., prostate cancer, psychological stress leading to impotence).

No official lists or scientific classifications of reproductive health hazards exist in the United States. Furthermore, although numerous research reports on specific agents contain findings that indicate harmful effects on human and/or animal reproductive systems and embryo/fetuses, these have not been systematically organized or used for purposes of occupational health policy or insurance analysis.

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[4] U.S. Chamber of Commerce, Analysis of Workers' Compensation Laws: 1982 (1982).

[5] See P. Barth, Workers' Compensation and Work-Related Illnesses and Diseases (1980) (8 percent); U.S. Department of Labor, Bureau of Labor Statistics, Supplementary Data System, table 121 (1980) (5 percent).

Table 9-1.—Summary of Occupational Disease Coverage by State

Nature of coverage[a]	Medical boards	Onset of disability or death	Time limit on claim filing	Medical care	Compensation[b]
Alabama: All diseases		Death—within 3 years after last exposure or last payment. Radiation or occupational pneumoconiosis*—exposure must occur in at least 12 months over 5 years prior to last exposure.	Disability—within 1 year after last exposure or last payment (radiation—within 1 year and claimant knows/should know relation to employment). Death— within 1 year after death or last payment. Coalminers pneumoconiosis—within 3 years after total disability or death and claimant knows/should know relation to employment.	Unlimited	Same as for accidents Coalminer's pneumoconiosis—total disability or death compensated same as Federal Black Lung Act.
Alaska: All diseases			2 years after knowledge of relation to employment. Within 1 year after death	Unlimited	Same as for accidents
American Samoa: All diseases	Claimant examined by physician selected by Commissioner.		Within 1 year after claimant knows/should know relation to employment.	Unlimited	Same as for accidents
Arizona: All diseases	Board of 3 medical consultants may be appointed by Commission. Report is prima facie evidence of facts.	Silicosis or asbestosis—employer liable only if exposure during 2 years.	Within 1 year after disability or accrual of right; excusable.*	Unlimited	Same as for accidents
Arkansas: All diseases		Disability or death—within 1 year after last exposure (3 years for silicosis or asbestosis), or 7 years for death following continuous disability. Does not apply to radiation. Silicosis or asbestosis presumed nonoccupational absent exposure in 5 years over 10 years prior to (disability & 2 of 5 years in-state unless same employer).	Disability—within 2 years after last exposure (silicosis or asbestosis—within 1 year from disablement; radiation—within 2 years from diagnosis). Death—within 2 years.	Unlimited	Same as for accidents. Silicosis and asbestosis—partial disability less than 33⅓ % noncompensable.*
California: All diseases. Special account for asbestos-related disease.			Disability—within 1 year from injury or last payment. Death—within 1 year after death (or death within 1 year of injury); 1 year after last medical payment; or 1 year after death if compensation paid; no proceedings more than 240 weeks after injury except for claims based on asbestos exposure.*	Unlimited	Same as for accidents
Colorado: All diseases		Disability—within 5 years after injury (no limit for radiation, asbestosis, silicosis, or anthracosis). Silicosis or asbestosis—employer liable only if exposure lasts 60 days.	Within 3 years after disability or death (5 years in case of ionizing radiation, asbestosis, silicosis, or anthracosis or if reasonable excuse).	Unlimited	Same as for accidents
Connecticut: All diseases	Panel of 3 physicians may be appointed by Commissioner to resolve medical issues involving lung disease.		Within 3 years after first manifestation of disease (within 2 years if death occurs within 2 years after first manifestation of disease, or 1 year after death, whichever is later).	Unlimited	Same as for accidents

[a] Employer and insurance carrier at time of last exposure are liable in Arkansas, Colorado, Florida, Georgia, Illinois, Indiana, Kansas, Kentucky, Maine, Maryland, New Hampshire, North Carolina, Oklahoma, Tennessee, Vermont, and Virginia. The employer at time of last exposure is liable in Alabama, Arizona, Iowa, Michigan, Missouri, Montana, New Mexico, Pennsylvania, South Dakota, Texas, and Utah. Liability is apportioned among responsible employers in New York and Rhode Island. California limits liability to employer during last year of exposure.

[b] Benefits determined as the date of last exposure or last injurious exposure in Arkansas, Georgia, Illinois, Indiana, Kentucky, Louisiana, Maine, Michigan, Minnesota, Missouri, New Jersey, South Dakota, Texas, Washington, Wisconsin, and Wyoming. Benefits determined as of the date of disability, knowledge, or manifestation in Alabama, Alaska, Arizona, California, Colorado, Connecticut, Delaware, Florida, Hawaii, Idaho, Iowa, Maryland, Massachusetts, Mississippi, Montana, Nebraska, New Hampshire, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Utah, Vermont, and West Virginia.

AL *Radiation illness caused by gradual exposure.
AZ *Limit on filing runs from when injury is manifest or when claimant knows/should know relation to employment; tolled during incapacity.
AK *Silicosis or asbestosis—worker who is affected but not disabled may leave work and receive up to 26 weeks of benefits plus up to $400 for retraining.
CA *Date of injury is date of disability and claimant knows/should know relation to employment.

Table 9-1.—Summary of Occupational Disease Coverage by State—Continued

Nature of coverage[a]	Medical boards	Onset of disability or death	Time limit on claim filing	Medical care	Compensation[b]
Delaware: All diseases			Disability or death—within 1 year after claimant knows relation to employment.	Unlimited	Same as for accidents
District of Columbia: All diseases			Within 1 year after injury, death, last payment, or knowledge of relation to employment.	Unlimited	Same as for accidents
Florida: All diseases		Death—following continuous disability and within 350 weeks after last exposure. Employer liable for dust disease only if exposure lasts 60 days.	Within 2 years after disablement, death, or last payment.	Unlimited	Same as for accidents
Georgia: All diseases	Medical board of 5, finding conclusive.	Within 1 year after last exposure (3 years for byssinosis, silicosis, or asbestosis; 7 years for death following continuous disability). Employer liable for silicosis or asbestosis only if exposure lasts 60 days, presumed nonoccupational absent exposure in 5 years over 10 years prior to disability (2 years in-state unless same employer).*	Within 1 year after disablement, death, or medical care, or 2 years after last payment.** Radiation—within 1 year after onset of disability and claimant knows/should know relation to employment.**	Unlimited	Same as for accidents***
Guam: All diseases			Within 1 year after injury, death, or last payment.	Unlimited	Same as for accidents
Hawaii: All diseases			Within 2 years after claimant knows relation to employment.	Unlimited	Same as for accidents
Idaho: All diseases		Within 1 year after last exposure (4 years for silicosis, 7 years for death following continuous disability). Employer liable for nonacute disease only if exposure lasts 60 days. Silicosis—exposure must occur in 5 years during 10 years prior to disablement (last 2 in-state unless same employer).	Within 1 year after manifestation or death. Silicosis—within 4 years after last exposure. Radiation or unusual disease—within 1 year after incapacity, disability, or death and claimant knows/should know relation to employment.	Unlimited	Same as for accidents. Silicosis—partial disability noncompensable.*
Illinois: All diseases		Disability—within 2 years after last exposure (3 years for berylliosis or silicosis, 25 years for asbestosis or radiation).	Disability—within 3 years after disablement or 2 years after last payment. Death—within 3 years after death or last payment. Coalminer's pneumoconiosis—within 5 years after last exposure or last payment.* Radiation—within 25 years after last exposure or 3 years after death or last payment.	Unlimited	Same as for accidents

[a] Employer and insurance carrier at time of last exposure are liable in Arkansas, Colorado, Florida, Georgia, Illinois, Indiana, Kansas, Kentucky, Maine, Maryland, New Hampshire, North Carolina, Oklahoma, Tennessee, Vermont, and Virginia. The employer at time of last exposure is liable in Alabama, Arizona, Iowa, Michigan, Missouri, Montana, New Mexico, Pennsylvania, South Dakota, Texas, and Utah. Liability is apportioned among responsible employers in New York and Rhode Island. California limits liability to employer during last year of exposure.

[b] Benefits determined as the date of last exposure or last injurious exposure in Arkansas, Georgia, Illinois, Indiana, Kentucky, Louisiana, Maine, Michigan, Minnesota, Missouri, New Jersey, South Dakota, Texas, Washington, Wisconsin, and Wyoming. Benefits determined as of the date of disability, knowledge, or manifestation in Alabama, Alaska, Arizona, California, Colorado, Connecticut, Delaware, Florida, Hawaii, Idaho, Iowa, Maryland, Massachusetts, Mississippi, Montana, Nebraska, New Hampshire, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Utah, Vermont, and West Virginia.

GA *Byssinosis claims diagnosed before July 1, 1983, must be filed before July 1, 1984.

**Year is 200 days exposure over 12 months.

***Silicosis or asbestosis—worker who is affected but not disabled may waive full compensation and if later disabled receive benefits for 100 weeks up to $2,000.

Table 9-1.—Summary of Occupational Disease Coverage by State—Continued

Nature of coverage[a]	Medical boards	Onset of disability or death	Time limit on claim filing	Medical care	Compensation[b]
Indiana: All diseases		Disablement—within 2 years after last exposure (3 years if caused by asbestos, coal, or silica dust); radiation—within 2 years after claimant knows/should know relation to employment. Death—within 2 years after disablement or during pendency of disability claim filed within that period; within 2 years after fixed disability expires but no later than 300 weeks after disablement. Employer liable for silicosis or asbestosis only if exposure lasts 60 days.	Within 2 years after disablement or death.	Unlimited	Same as for accidents
Iowa: All diseases	Medical board may decide controverted medical questions or provide medical examinations for certain employees.	Disability or death—within 1 year after last exposure (3 years for pneumoconiosis; 7 years for death following continuous disability). Pneumoconiosis presumed nonoccupational absent exposure in 5 years over 10 years prior to disability (2 of 5 years in-state); employer liable only if exposure lasts 60 days.	Within 2 years after death or disablement or 3 years after last payment. Radiation—within 90 days after disablement or death and claimant knows/should know relation to employment.	Unlimited	Same as for accidents. Pneumoconiosis—partial disability less than 33⅓% is noncompensable.
Kansas: All diseases		Disability or death—within 1 year after last exposure (3 years for death from silicosis, 7 years for death following continuous disability). Does not apply to radiation. Silicosis presumed nonoccupational absent exposure in 5 years over 10 years prior to disability (2 of 5 years in-state unless same employer); employer liable only if exposure lasts 60 days.	Within 1 year after disablement, death, or last payment (2 years after last payment in case of silicosis). Radiation—within 1 year after claimant knows/should know relation to employment.	Unlimited	Same as for accidents*
Kentucky: All diseases*			Disability—within 3 years after last exposure or first manifestation. Death—within 3 years, if it occurs within 3 years after last exposure or first manifestation. Limit waived where voluntary payment or employer knows of disease and cause. No claim more than 5 years after last exposure (20 years in case of radiation), except for death within 20 years after continuous disability begins in cases where there is award or timely claim for disability.	Unlimited	Same as for accidents. Where disablement occurs after 5 years exposure or results from silicosis or pneumoconiosis, apportioned between employer and Special Fund. Fund pays 75% of cost if not conclusively proven to result from last exposure, otherwise pays 40%. Employer pays balance.
Louisiana: All diseases		Diseases contracted in less than 1 year presumed to be nonoccupational. Presumption is rebuttable by "overwhelming preponderance of evidence."	Disability—within 6 months after manifestation, occurrence of disability, or worker knows/should know relation to employment. Death—within 6 months, or within 6 months after worker knows/should know relation to employment.	Unlimited	Same as for accidents

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[a] Employer and insurance carrier at time of last exposure are liable in Arkansas, Colorado, Florida, Georgia, Illinois, Indiana, Kansas, Kentucky, Maine, Maryland, New Hampshire, North Carolina, Oklahoma, Tennessee, Vermont, and Virginia. The employer at time of last exposure is liable in Alabama, Arizona, Iowa, Michigan, Missouri, Montana, New Mexico, Pennsylvania, South Dakota, Texas, and Utah. Liability is apportioned among responsible employers in New York and Rhode Island. California limits liability to employer during last year of exposure.

[b] Benefits determined as the date of last exposure or last injurious exposure in Arkansas, Georgia, Illinois, Indiana, Kentucky, Louisiana, Maine, Michigan, Minnesota, Missouri, New Jersey, South Dakota, Texas, Washington, Wisconsin, and Wyoming. Benefits determined as of the date of disability, knowledge, or manifestation in Alabama, Alaska, Arizona, California, Colorado, Connecticut, Delaware, Florida, Hawaii, Idaho, Iowa, Maryland, Massachusetts, Mississippi, Montana, Nebraska, New Hampshire, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Utah, Vermont, and West Virginia.

KS *Worker who is affected but not disabled may waive full compensation and if later disabled receive benefits up to 100 weeks.

KY *Black lung claimant must file under state and federal law.

Table 9-1.—Summary of Occupational Disease Coverage by State—Continued

Nature of coverage[a]	Medical boards	Onset of disability or death	Time limit on claim filing	Medical care	Compensation[b]
Maine:					
All diseases		Incapacity—within 3 years after last exposure (does not apply to asbestos-related disease). Employer liable only if exposure lasts 60 days (except for radiation and asbestos-related disease). Silicosis presumed nonoccupational absent in-state exposure in 2 years during 15 years preceding disability (part of exposure may be out of state if same employer).	Within 2 years after incapacity or 1 year after death or last payment (40 years after last payment for asbestos-related disease).* If mistake of fact, within reasonable time but no later than 10 years after last payment. Radiation—limit runs from date of incapacity and claimant knows/should know relation to employment.	Unlimited	Same as for accidents
Maryland:					
All diseases			Within 2 years after disablement, death, or actual knowledge of relation to employment, excusable (3 years for pulmonary dust disease).	Unlimited	Same as for accidents
Massachusetts:					
All diseases			Within 1 year after injury or death; excusable.	Unlimited	Same as for accidents
Michigan:					
All diseases			Within 2 years after claimant knows/should know relation to employment.	Unlimited	Same as for accidents
Minnesota:					
All diseases			Within 3 years after employee's knowledge of cause of injury or disability.	Unlimited	Same as for accidents
Mississippi:					
All diseases			Within 2 years after injury* or death.	Unlimited	Same as for accidents
Missouri:					
All diseases			Within 2 years after injury, death, or last payment (3 years if no injury report filed); limitation runs from date injury is reasonably apparent.	Unlimited	Same as for accidents
Montana:					
All diseases	Examinations made by 1 or more members of the occupational disease panel.	Death—within 3 years after last employment unless continuous total disability (does not apply to radiation). Silicosis—total disability or death must occur within 3 years after last employment (except for death following continuous total disability), and employer is liable only if exposure lasts 90 workshifts.*	Within 1 year after disability and claimant knows/should know relation to employment; may be extended 2 more years. No claim more than 3 years after last employment (except for radiation or death after continuous total disability).	Unlimited	Same as for accidents, excluding partial disability. Worker who is affected but not disabled may leave job and receive compensation up to $10,000. Pneumoconiosis benefits reduced by amount payable under federal law. Benefits for silicosis are supplemented so that combined compensation is $200 monthly; supplement is general revenue financed.
Nebraska:					
All diseases			Within 2 years after injury or death.	Unlimited	Same as for accidents

[a] Employer and insurance carrier at time of last exposure are liable in Arkansas, Colorado, Florida, Georgia, Illinois, Indiana, Kansas, Kentucky, Maine, Maryland, New Hampshire, North Carolina, Oklahoma, Tennessee, Vermont, and Virginia. The employer at time of last exposure is liable in Alabama, Arizona, Iowa, Michigan, Missouri, Montana, New Mexico, Pennsylvania, South Dakota, Texas, and Utah. Liability is apportioned among responsible employers in New York and Rhode Island. California limits liability to employer during last year of exposure.

[b] Benefits determined as the date of last exposure or last injurious exposure in Arkansas, Georgia, Illinois, Indiana, Kentucky, Louisiana, Maine, Michigan, Minnesota, Missouri, New Jersey, South Dakota, Texas, Washington, Wisconsin, and Wyoming. Benefits determined as of the date of disability, knowledge, or manifestation in Alabama, Alaska, Arizona, California, Colorado, Connecticut, Delaware, Florida, Hawaii, Idaho, Iowa, Maryland, Massachusetts, Mississippi, Montana, Nebraska, New Hampshire, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Utah, Vermont, and West Virginia.

ME *Claim for asbestos-related disease contracted between 11/30/67 and 10/1/83 must be filed by 1/1/85.

MD *Disease or injury compensable under federal law (other than Social Security Disability Insurance) is not compensable.

MI *Silicosis, dust disease, and logging industry fund reimburses compensation over $12,500 (expires 1/1/86).

MS *For radiation, date of disablement is date of injury.

MT *Silicosis is noncompensable absent in-state exposure in 1,000 workshifts during 8 years preceding total disability; claimant who is discharged to avoid liability may receive compensation when totally disabled if employed 700 workshifts.

Table 9-1.—Summary of Occupational Disease Coverage by State—Continued

Nature of coverage[a]	Medical boards	Onset of disability or death	Time limit on claim filing	Medical care	Compensation[b]
Nevada: All diseases	Physician review board selected by insurer; findings conclusive.	Silicosis or respiratory dust disease is noncompensable absent in-state exposure in 3 years during 10 years preceding disability or death.	Within 90 days after knowledge of disability and relation to employment or 1 year after death. Silicosis or respiratory dust disease—within 1 year after temporary or total disability or death.	Unlimited	Same as for accidents
New Hampshire: All diseases			Within 2 years after injury or death and claimant knows/should know of injury and relation to employment.*	Unlimited	Same as for accidents
New Jersey: All diseases			Within 2 years after claimant knows relation to employment or last payment.	Unlimited	Same as for accidents
New Mexico: All diseases		Death—within 1 year after last employment (3 years for death following continuous disability), and death must follow disability within 2 years. Silicosis or asbestosis—disability or death within 2 years after last employment (5 years for death following continuous disability); employer is liable only if exposure lasts 60 days; noncompensable absent in-state exposure in 1,250 workshifts during 10 years preceding disability. Radiation—disability or death within 10 years after last employment.	Within 1 year after disability or death or 1 year 31 days after last voluntary payment. Radiation—within 1 year after disability begins or death and claimant knows/should know relation to employment.	Unlimited	Same as for accidents
New York: All diseases		Disease must be contracted within 12 months prior to disablement except in case of continuous employment for one employer or in case of certain illnesses* (within 5 years prior to death if no claim prior to death in case of certain illnesses*). Not applicable to radiation, silicosis, or other dust diseases.	Within 2 years after disablement or death. Radiation, silicosis, or dust disease—within 90 days after disablement and claimant knows relation to employment.	Unlimited	Same as for accidents**
North Carolina: All diseases	Commission appoints 3-member advisory board for silicosis or asbestosis cases.	Death within 2 years after injury; if totally disabled 6 years after injury or 2 years after final determination. Asbestosis—disability or death within 10 years after last exposure; for death following continuous disability, disability must occur within 10 years after last exposure.* Lead poisoning—disability or death within 2 years after last exposure; for death following continuous disability, disability must occur within 2 years after last exposure.	Within 2 years after disablement, death, or last payment. Radiation—within 2 years after incapacity and claimant knows/should know relation to employment. Brown lung claims compensable regardless of last exposure, effective 6/25/80-4/30/81.	Unlimited	Same as for accidents**

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[a] Employer and insurance carrier at time of last exposure are liable in Arkansas, Colorado, Florida, Georgia, Illinois, Indiana, Kansas, Kentucky, Maine, Maryland, New Hampshire, North Carolina, Oklahoma, Tennessee, Vermont, and Virginia. The employer at time of last exposure is liable in Alabama, Arizona, Iowa, Michigan, Missouri, Montana, New Mexico, Pennsylvania, South Dakota, Texas, and Utah. Liability is apportioned among responsible employers in New York and Rhode Island. California limits liability to employer during last year of exposure.

[b] Benefits determined as the date of last exposure or last injurious exposure in Arkansas, Georgia, Illinois, Indiana, Kentucky, Louisiana, Maine, Michigan, Minnesota, Missouri, New Jersey, South Dakota, Texas, Washington, Wisconsin, and Wyoming. Benefits determined as of the date of disability, knowledge, or manifestation in Alabama, Alaska, Arizona, California, Colorado, Connecticut, Delaware, Florida, Hawaii, Idaho, Iowa, Maryland, Massachusetts, Mississippi, Montana, Nebraska, New Hampshire, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Utah, Vermont, and West Virginia.

NH *Date of injury is last date of exposure or first date worker knows/should know relation to employment.

NY *Illness caused by compressed air, arsenic, benzol, beryllium, zirconium, cadmium, chrome, lead, or fluorine.

**Disability or death due to silicosis or dust disease reimbursed from special fund for all payments over 104 weeks.

NC *Asbestosis is noncompensable absent in-state exposure in 2 years during 10 years preceding last exposure or if exposure is less than 30 working days in 7 consecutive months.

**Worker who is affected but not disabled by asbestosis or silicosis or who is removed from exposure receives benefits up to $80 weekly for 104 weeks. If later totally disabled, full compensation is paid. If death results within 2 years after last exposure (350 weeks if caused by secondary infection), full compensation is paid. If partially disabled 66⅔% of wage loss is paid for another 196 weeks. If unrelated death, balance of 104 weeks is paid plus 300 weeks (total disability) or percentage of 196 weeks (partial disability). Worker may waive full compensation and receive 104 weeks of compensation plus 100 more weeks if later disabled or dies.

Table 9-1.—Summary of Occupational Disease Coverage by State—Continued

Nature of coverage[a]	Medical boards	Onset of disability or death	Time limit on claim filing	Medical care	Compensation[b]
North Dakota:					
All diseases		Death—within 1 year after injury if no disability, or 1 year after cessation of disability, or 6 years after injury if disability is continuous.*	Within 1 year after injury; within 2 years after death (2 years after injury if no claim prior to death).*	Unlimited	Same as for accidents
Ohio:					
All diseases	Medical specialist in specific cases; findings advisory.		Within 2 years after disability or death or within 6 months after diagnosis (whichever is later).	Unlimited	Same as for accidents. No partial disability for respiratory dust disease.*
Oklahoma:					
All diseases		Employer liable for silicosis or asbestosis only if exposure lasts 60 days.	Within 18 months after last exposure or manifestation and diagnosis by a physician, or within 3 months after disablement.	Unlimited	Same as for accidents*
Oregon:					
All diseases			Within 5 years after last exposure and within 180 days after disablement or physician informs claimant of disablement 10 years after last exposure for radiation disease.*	Unlimited	Same as for accidents
Pennsylvania:					
All diseases	Examination by impartial physician may be ordered.	Within 300 weeks after last exposure (except death following disability that occurs within 300 weeks after last exposure). Silicosis, anthracosilicosis, or coalminer's pneumoconiosis—noncompensable absent in-state exposure in 2 years during 10 years preceding disability.*	Within 3 years after disablement, death, or last payment. Radiation—within 3 years after the employee knows/should know relation to employment.	Unlimited	Same as for accidents*
Puerto Rico:					
Diseases as provided by law		Disability—within 1 year after last exposure, except diseases with longer latency periods.	Within 3 years from time employee learns nature of disability.	Unlimited	Same as for accidents
Rhode Island:					
All diseases	Director of Labor appoints impartial physician.		Within 3 years after disability or death. Radiation—within 1 year after claimant knows/should know relation to employment.	Unlimited	Same as for accidents
South Carolina:					
All diseases	Medical board determines controverted medical questions; pulmonary cases may be referred to pulmonary specialists of state medical universities.	Disease must be contracted within 1 year after last exposure (2 years for pulmonary dust disease), except radiation. Byssinosis is noncompensable absent exposure for 7 years.	Within 2 years after definitive diagnosis or 1 year after death. Radiation—limitation runs from date of disability and claimant knows/should know relation to employment	Unlimited	Same as for accidents. Worker who is affected but not disabled may waive compensation (except radiation).

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[a] Employer and insurance carrier at time of last exposure are liable in Arkansas, Colorado, Florida, Georgia, Illinois, Indiana, Kansas, Kentucky, Maine, Maryland, New Hampshire, North Carolina, Oklahoma, Tennessee, Vermont, and Virginia. The employer at time of last exposure is liable in Alabama, Arizona, Iowa, Michigan, Missouri, Montana, New Mexico, Pennsylvania, South Dakota, Texas, and Utah. Liability is apportioned among responsible employers in New York and Rhode Island. California limits liability to employer during last year of exposure.

[b] Benefits determined as the date of last exposure or last injurious exposure in Arkansas, Georgia, Illinois, Indiana, Kentucky, Louisiana, Maine, Michigan, Minnesota, Missouri, New Jersey, South Dakota, Texas, Washington, Wisconsin, and Wyoming. Benefits determined as of the date of disability, knowledge, or manifestation in Alabama, Alaska, Arizona, California, Colorado, Connecticut, Delaware, Florida, Hawaii, Idaho, Iowa, Maryland, Massachusetts, Mississippi, Montana, Nebraska, New Hampshire, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Utah, Vermont, and West Virginia.

ND *Date of injury is date on which a reasonable person knows/should know relation to employment.

OH *Includes asbestosis, silicosis, and coalminer's pneumoconiosis. Worker who is affected but not disabled by respiratory dust disease and leaves employment may receive $49 weekly for 30 weeks, 66⅔% of wage loss (not to exceed $40.25 weekly).

OK *Worker who is affected but not disabled by silicosis or asbestosis may waive compensation for aggravation of disease and, if later disabled, receive benefits for 100 weeks up to $2,000.

OR *Asbestos-related disease—within 40 years after last exposure and 180 days after disability or knowledge of disability.

PA *Under Occupational Disease Act, State pays $125 monthly for total disability or death caused by silicosis, anthracosilicosis, coalminer's pneumoconiosis, or asbestosis, provided there has been 2 years of in-state exposure, in cases where the claim is barred by the statute of limitations and the last exposure occurred before 1965 or where exposure occurred under several employers.

Table 9-1.—Summary of Occupational Disease Coverage by State—Continued

Nature of coverage[a]	Medical boards	Onset of disability or death	Time limit on claim filing	Medical care	Compensation[b]
South Dakota: All diseases	Division may contract with physicians for reports.	Silicosis—noncompensable absent in-state exposure in 2 years (in-state requirement waived if same employer); employer liable only if exposure lasts 60 days.	Within 2 years after disability or death. Radiation—within 1 year after disability and claimant knows relation to employment.	Unlimited	Same as for accidents. No permanent partial disability for silicosis.*
Tennessee: All diseases			Within 1 year after incapacity or death.	Unlimited	Same as for accidents. Coalminer's pneumoconiosis—same as Federal Black Lung Act.
Texas: All diseases	Provides for medical committee to pass on controverted questions and with power to order examinations.		Within 1 year after injury or first distinct manifestation, 1 year after death. May be extended.		Same as for accidents
Utah: All diseases	Commission appoints medical panel of 1 or more to report on extent of disability.	Partial disability—within 2 years after last exposure. Total disability—within 1 year after last employment; for silicosis, 3 years (uncomplicated) or 5 years (complicated). Death—within 3 years after last employment (5 years for complicated silicosis or death following continuous total disability). Not applicable to radiation. Silicosis—noncompensable absent 5 years in-state exposure in 15 years preceding disability; employer liable only if exposure lasts 30 days.	Within 1 year after incapacity or death and claimant knows/should know relation to employment, but no later than 3 years after death. Permanent partial disability—within 2 years.	Unlimited	Same as for accidents*
Vermont: All diseases		Disablement—within 5 years after last exposure. Death—during employment or after continuous disability beginning within 5 years after last exposure, but no later than 12 years after last exposure. Does not apply to radiation.	Within 1 year after discovery, death, or last payment. Radiation—within 1 year after first incapacity and worker knew/should have known relation to employment.	Unlimited	Same as for accidents. Affected but nondisabled worker may waive full compensation and later receive limited compensation.
Virgin Islands: All diseases			Within 60 days after disability.	Unlimited	Same as for accidents
Virginia: All diseases		Exposure in 90 workshifts conclusively presumed injurious exposure.	Within 2 years after diagnosis is first communicated to worker, or within 5 years after last exposure, whichever is first.* Within 3 years after death occurring within periods for disability.	Unlimited	Same as for accidents. Worker who is affected but not disabled may waive compensation.
Washington: All diseases			Within one year after physician's notice to claimant.	Unlimited	Same as for accidents
West Virginia: All diseases	Occupational Pneumoconiosis Board appointed by Commissioner determines medical questions.	Occupational pneumoconiosis is noncompensable absent 2 years continuous in-state exposure in 10 years before last exposure or exposure in 5 years during the 15 years before last exposure.	Within 3 years after knowledge or last exposure. Within 2 years after death.	Unlimited	Same as for accidents

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[a] Employer and insurance carrier at time of last exposure are liable in Arkansas, Colorado, Florida, Georgia, Illinois, Indiana, Kansas, Kentucky, Maine, Maryland, New Hampshire, North Carolina, Oklahoma, Tennessee, Vermont, and Virginia. The employer at time of last exposure is liable in Alabama, Arizona, Iowa, Michigan, Missouri, Montana, New Mexico, Pennsylvania, South Dakota, Texas, and Utah. Liability is apportioned among responsible employers in New York and Rhode Island. California limits liability to employer during last year of exposure.

[b] Benefits determined as the date of last exposure or last injurious exposure in Arkansas, Georgia, Illinois, Indiana, Kentucky, Louisiana, Maine, Michigan, Minnesota, Missouri, New Jersey, South Dakota, Texas, Washington, Wisconsin, and Wyoming. Benefits determined as of the date of disability, knowledge, or manifestation in Alabama, Alaska, Arizona, California, Colorado, Connecticut, Delaware, Florida, Hawaii, Idaho, Iowa, Maryland, Massachusetts, Mississippi, Montana, Nebraska, New Hampshire, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Utah, Vermont, and West Virginia.

SD *Worker who is affected by silicosis but not disabled may waive full compensation and if later disabled or dies receive benefits up to $2,000; if leaves employment, may receive compensation up to $1,000.

UT *Worker with permanent partial disability who must change occupation may receive up to $1,000 for vocational rehabilitation and retraining, plus compensation of 66⅔% of average weekly wages up to 66⅔% of SAWW for up to 20 weeks, then additional compensation (cumulative total may not exceed $2,080).

VA *5-year limitation does not apply to cataract of the eyes, skin cancer, radium disability, ulceration, undulant fever, angiosarcoma of the liver due to vinyl chloride exposure, or mesothelioma; byssinosis—within 7 years after last exposure; coalminer's pneumoconiosis—within 3 years after diagnosis; asbestosis—within 2 years after diagnosis (if based on changed condition, within 2 years after diagnosis of advanced stage).

Table 9-1.—Summary of Occupational Disease Coverage by State—Continued

Nature of coverage[a]	Medical boards	Onset of disability or death	Time limit on claim filing	Medical care	Compensation[b]
Wisconsin: All diseases	May appoint independent medical expert in doubtful cases.		Unlimited. After 12 years claim may be filed with state fund.	Unlimited	Same as for accidents
Wyoming: All diseases	Yes		Within 1 year after diagnosis or 3 years after exposure, whichever is last. Radiation—within 1 year after diagnosis or death.	Unlimited	Same as for accidents
F.E.C.A.: All diseases			Within 3 years after injury, death, or disability and claimant knows/should know relation to employment; excusable.	Unlimited	Same as for accidents
Longshore Act: All diseases			Within 1 year after injury, death, last payment, or knowledge of relation to employment.	Unlimited	Same as for accidents

[a] Employer and insurance carrier at time of last exposure are liable in Arkansas, Colorado, Florida, Georgia, Illinois, Indiana, Kansas, Kentucky, Maine, Maryland, New Hampshire, North Carolina, Oklahoma, Tennessee, Vermont, and Virginia. The employer at time of last exposure is liable in Alabama, Arizona, Iowa, Michigan, Missouri, Montana, New Mexico, Pennsylvania, South Dakota, Texas, and Utah. Liability is apportioned among responsible employers in New York and Rhode Island. California limits liability to employer during last year of exposure.

[b] Benefits determined as the date of last exposure or last injurious exposure in Arkansas, Georgia, Illinois, Indiana, Kentucky, Louisiana, Maine, Michigan, Minnesota, Missouri, New Jersey, South Dakota, Texas, Washington, Wisconsin, and Wyoming. Benefits determined as of the date of disability, knowledge, or manifestation in Alabama, Alaska, Arizona, California, Colorado, Connecticut, Delaware, Florida, Hawaii, Idaho, Iowa, Maryland, Massachusetts, Mississippi, Montana, Nebraska, New Hampshire, New Mexico, New York, North Carolina, North Dakota, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, Tennessee, Utah, Vermont, and West Virginia.

SOURCE: U.S. Chamber of Commerce, Analysis of Workers Compensation Laws, 1984.

The reviews of the scientific literature that have been published[6] are incomplete in two ways. First, there is a lack of exhaustive research about the effects on reproductive function of most chemical, physical, and biological agents. The information available about a particular exposure is limited by the number and quality of animal and human studies of various aspects of exposure (e.g., dose, time, response). (See chapter 4.) Second, published sources do not reflect unpublished studies carried out in the private sector or by government agencies.

Thus no medical or scientific structuring of reproductive health hazards (or of occupational disease problems generally)—either by agent, occupational classification, or type of victim (male or female adult, embryo/fetus)—is currently available to guide either the workers' compensation system or legislators who have the power to improve the system. As a result, the compensation system in each State proceeds on a case-by-case basis with various types of reproductive injury or disease claims.

Criteria for Securing Benefits for Reproductive Harms[7]

In most States, workers' compensation is viewed as a system enacted primarily for the benefit of employees, and the various boards, courts, and legislatures broadly construe the relevant law to promote the accomplishment of its beneficent design. A major reason for this "beneficent" view is the harsh reality that each State's workers' compensation law provides that it constitutes the exclusive remedy for the injured or diseased worker, and thereby abrogates the common law rights of the worker against the employer for wrongful acts. The low level of compensation available also induces liberal construction of the workers' compensation law, since not much money is at stake in any individual claim. Finally, even boards and courts that do not view the system as beneficent nevertheless tend to construe workers' compensation laws liberally, since the alternative would be to force claims that fail into the tort system, thereby exposing industry to much higher economic risks and severely crowding court dockets. Nevertheless, reproductive harms will not generally satisfy the criteria for compensability even if the criteria are liberally construed.

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[6] S. Barlow and F. Sullivan, Reproductive Hazards of Industrial Chemicals (1982); Guidelines for Studies of Human Populations Exposed to Mutagenic and Reproductive Hazards (A. Bloom (ed.) (1981); III Assessment of Reproductive and Teratogenic Hazards (M. Christian, W. Galbraith, P. Voytek, and M. Mehlman (eds.) (1983); I. Nisbet and N. Karch, Chemical Hazards to Human Reproduction (1983)).

[7] A. Larson, The Law of the Workmen's Compensation § 65.20 (1978).

To secure workers' compensation for an injury or disease, a claimant must meet several legal requirements. (Criteria differ among States; only their general features are discussed here.) **There are three major requirements, common to most if not all State compensation systems, that affect a worker's ability to secure benefits for reproductive harms caused by workplace exposures. These are the requirement of a *"personal" injury or disease,* that the injury or disease result in *job disability,* and that the injury or disease be caused by a *workplace accident or exposure.***

"Personal" Injury or Disease

At the outset of the claims process in all States, the worker needs evidence of diagnosis of a "personal" injury or disease. This requirement precludes compensation for injuries or diseases suffered by others, such as the worker's spouse, fetus, child, or descendant. Thus, if the condition is job-related and impairs the male worker's ability to cause conception (e.g., by causing impotency, infertility, or sterility) or the female worker's ability to conceive and carry a fetus to term (e.g., infertility, sterility, spontaneous abortion, or miscarriage), the disease or injury is considered personal to the worker and is eligible for compensation so long as it meets the various other criteria discussed later. **In most States, the personal injury criterion constitutes a barrier to claims for reproductive harms that involve the developing offspring,** including birth defects, decreased birth weight, change in gestational age at delivery, altered sex ratio, multiple births, infant death, and childhood morbidity or mortality (see table 9-2). It is important to note, however, that the worker's spouse, embryo/fetus, offspring, and descendants may be able to sue the employer under tort law principles (discussed in chapter 10).

Disability

Claims for reproductive harms that survive the "personal" injury test and satisfy various procedural requirements must still overcome other obstacles. One is the requirement that the claimant is disabled or otherwise qualifies for some type of benefits (e.g., benefits for loss of bodily function that do not result in disability if provided for by a State benefit scheme).

State workers' compensation laws vary, but typically provide for several different classes of benefits and set forth the proof that is needed to qualify for such benefits. The most common types of benefits are those for job disability or loss of earnings, medical costs, death, and bur-

Table 9-2.—Eligibility for Compensation for Reproductive Harms: Personal Injury Criterion

	Victim			
Circumstances of harm	Worker	Spouse	Embryo/fetus	Offspring
1. Accidental injury to worker reproductive system or fetus	Eligible	Not eligible	Not eligible	Not eligible
2. Physical stress on worker	Eligible	Not eligible	Not eligible	Not eligible
3. Acute or chronic exposure of worker's reproductive system, or of fetus, spouse, or offspring	Eligible	Not eligible	Not eligible	Not eligible
4. "Side effect" cases (worker reproductive function impaired due to other injuries or diseases)	Eligible, but "other" injury or disease will be primary personal injury for compensation purposes, not reproductive injury	Not eligible	Not eligible	Not eligible

NOTE: "Personal" injuries include: sexual dysfunction (libido, potency), sperm and ovum abnormalities, infertility, illness during pregnancy and parturition, early and late fetal loss, and worker's age at menopause. Personal injuries do not include any injury to any person other than the worker (e.g., spouse, fetus that results in offspring, or offspring).

SOURCE: Office of Technology Assessment.

ial. A number of States also provide modest benefits for a few specified losses of bodily functions. Job disability benefits are the most important form of compensation because they provide for support of the worker and his or her family over an extended period of time. The dollar levels for disability compensation tend to be low for two reasons: they are adjusted infrequently by the various State legislatures; and benefit levels are usually based on a predetermined percentage of the worker's wages, often the wages at the time of exposure rather than the wages at the time the disability begins. Since years may elapse between the time of exposure and the manifestation of an occupational disease, benefits may be substantially lower for an occupational disease victim than for an occupational injury victim who has been similarly disabled. Some States have adopted automatic cost-of-living adjustments as a remedy for these problems.

A reproductively harmed worker can generally recover medical benefits for incurred medical expenses if his or her medical problem meets the personal injury criterion discussed earlier and the worker can prove the job-relatedness of the injury. **In many, if not most, cases of reproductive injury or disease, medical benefits alone are inadequate because they are not designed to compensate for a temporary or permanent loss of sexual and reproductive function, only to compensate for medical treatment costs. But unless the worker is disabled, he or she will often not be able to collect a monetary substitute for lost or diminished sexual or reproductive functions under the workers' compensation system.**

Up to four subclasses of job disability benefits are provided in some States: temporary-total, temporary-partial, permanent-total, and permanent-partial. For total disability benefits, the worker must be incapable of earning wages or performing any work for compensation. For partial disability benefits, the worker may be able to work, but must be unable to earn his or her former average wage in order to be eligible to receive the differential amount between past and present wage levels unless a schedule covers the injury. Either type of benefit may be received for as long as the worker is disabled, temporarily or permanently. However, both partial and total disability benefits are subject to legislatively imposed limits that generally keep the disabled worker's total income at or below the average statewide or nationwide industrial wage. In addition to these benefits, the disabled worker may be entitled to secure benefits from other private and public compensation systems (e.g., Social Security disability benefits or private insurance disability benefits).

The requirement of a disability generally prevents the award of disability benefits for most claims of reproductive injury or disease, since such harms do not usually disable the worker or prevent him or her from resuming work at the same job. Of the few reproductive endpoints that meet the personal injury criterion (see table 9-2), only occupationally caused injury to reproductive organs, illness during pregnancy, and fetal loss are likely to result in any job disability, and this will usually be temporary disability at most. When a reproductive harm is sufficiently disabling to prevent the employee from performing the job for a temporary period, as in the case of a job-induced miscarriage, the worker is entitled to collect disability benefits. However, even when workers are able to make a connection between a workplace exposure and their disability, the short duration of the disability period makes such workers much more likely to take advantage of employer-provided sick leave and health insurance benefits than face the expense, risk of the claim being denied, loss of medical privacy, and low benefits endemic to workers' compensation claims. Thus, although disability compensation is *theoretically* available to a small number of reproductively harmed workers, they are unlikely to claim this entitlement. In those States that permit compensation for the loss of a bodily function, and where bodily function has been construed to include reproductive function, however, claims for reproductive harms that meet the "personal injury" test, detailed in table 9-2, may be compensable even when not occupationally disabling.

Some States provide a special benefit category for disfigurement or loss of function, which may include sexual or reproductive function, without requiring disability. Workers' compensation officials in 10 States reported to OTA that they would award compensation over and above medical benefits for disfigurement or loss of some bodily function, while 9 reported they would pay medical benefits only, and 7 indicated no source of compensation.[8] It should be noted, however, that most of the States that compensate for nondisabling reproductive injuries generally do so only for a narrow class of injuries that are listed for a scheduled benefit, such as cases of testicular injury or loss. Only a few States, such as North Carolina, provide benefits for a broader range of reproductive disorders. The North Carolina Workers' Compensation Act provides that:

> In case of the loss of or permanent injury to any important external or internal organ or part of the body for which no compensation is payable under any other subdivision of this section, the Industrial Commission may award proper and equitable compensation not to exceed ten thousand dollars.[9]

Job-Relatedness (Causation)

Causation evidence is required in each State's compensation system, because the governing statutes typically require that compensation coverage and benefits apply only to claims "arising out of and in the course of employment." Usually the claimant has the burden of proof to persuade the compensation board that the claim is based on an *occupational* injury or disease. Causation as a determinant of eligibility for compensation assures that legislative purposes will be met, and that the system will not be abused by spurious claims that would impose additional costs on employers, their insurers, and ultimately the public.

In practice, a board's threshold for causation evidence is relatively low, compared to the evidentiary requirements in common law litigation, because of the previously mentioned view of the workers' compensation system as "beneficent." This attitude of beneficence is reinforced by the relatively low level of compensation and the harshness of the exclusivity of remedy rule (discussed later). Thus, from the perspective of the boards in many States, it serves neither worker nor employer interests to use stringent, claim-denying criteria for causation evidence. Nevertheless, **a worker who has the type of reproductive problem that also occurs from nonoccupational causes (e.g., sexual dysfunction, infertility, spontaneous abortion) may have a real problem proving that his or her problem resulted from an exposure at work.**

Level of proof requirements differ among the States, but fall into several general categories, with "preponderance of the evidence" being the most common. This standard requires evidence to establish that the particular disease be more likely to have been caused by a workplace exposure than by some other cause. Some States have more stringent tests such as "must be clearly proven." Proof generally consists of written reports and/or oral testimony by medical professionals who have examined the claimant and, perhaps, reviewed the medical literature. The credibility of the doctor as expert will often be a key issue in contested disease claims of complex etiology. When a doctor's evidence alone is inadequate to support a finding of job-relatedness for a disease of complex origins, disease claims may also require oral or written testimony by toxicologists, epidemiologists, biologists, medical researchers, and other scientific experts. Even scientific experts may be unable to persuade a board that they have a reasonable certainty as to the cause of a worker's cancer, sterility, miscarriage, or other health problem. Epidemiological evidence is given weight in some States, toxicological evidence is generally accorded less significance, and neither type of evidence is likely to be considered as important as medical evidence by a physician.

Over time, as clusters of claims for certain types of diseases emerge, boards gain familiarity with these diseases, and causal relationships may be more easily established. When these events occur, State legislatures sometimes respond by setting forth minimal evidentiary requirements for claimants with such diseases. Experience with clusters has thus led to numerous statutory (and in some cases, judicial) modifications setting forth abbreviated evidentiary requirements for nonreproductive diseases such as black lung, asbestosis, and silicosis. Several States have taken the further step of establishing medical review panels or permitting the use of board-appointed physicians to assist their compensation boards.

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[8] Twenty-six States responded to the survey.

[9] N.C. Gen. Stat. § 97-31(24) (1979).

Evidence of causation has always been one of the critical legal issues in workers' compensation law. It has become even more critical and controversial because of the rising incidence and importance of disease claims and the new types of disease claims that have complex etiology. Some of the most troublesome issues arise from the different perspectives on causation held by doctors, scientists, and lawyers, as well as by the courts and legislators. Doctors are trained to diagnose, not to establish causation for, individual cases. Scientific views of causation involve considerations of multiple etiologic factors, and analysis of their interactions based on population studies, animal tests, and in vitro studies. The legal view stresses whether a particular event or element was the proximate, precipitating cause, often to the exclusion of other factors and their interactions. While the medical and scientific views emphasize pre-existing and extra-workplace conditions (e.g., prior work exposure, genetics, lifestyle), the legal view commonly holds that all events occurring prior to or apart from the employment at issue are irrelevant, and the worker's existing medical and non-job-related vulnerabilities are taken as a given.

To the physician or scientist, proof means virtual certainty, a probability in the 95 to 99 percent range, whereas the law merely requires proof that the allegation is more likely true than false, a 51 percent probability. Thus:

> . . . for the occupational disease claimant the burden of proving causability . . . becomes prohibitive when, as is often the case, medical experts can at best venture a guess, or testify to a probability that a particular . . . disease is in fact employment related. Epidemiological studies demonstrating a high probability of employment-relatedness of lung cancer in an asbestos insulation worker, for example, would probably not establish causation in an individual claim.[10]

Although compensation board attitudes today are perhaps more liberal towards the admissibility and weight to be accorded scientific evidence, particularly statistical or epidemiological evidence of a probabilistic nature, the boards are also cautious, skeptical, and inconsistent. Therefore, despite their beneficent view, boards generally still prefer medical evidence that a particular individual contracted a particular disease in a particular way, to scientific evidence that shows how many, or even most, people contract the disease. Both workers claiming benefits for occupational disease and insurers defending against such claims are unhappy with this situation and believe that a more receptive approach by boards and courts would work to the advantage of their differing interests.[11]

Defendants (insurers and employers) disputing disease claims frequently argue that the claimant failed to establish the necessary causal relationship. In such cases, defendants may gain a distinct advantage from a more receptive approach to epidemiological and other scientific evidence. Defendants would then be able to use statistical evidence to better dispute claims on grounds of the complex etiology of disease, pre-existing disability (in States where such evidence is useful), conflicting studies and results, and intervening causes that were not job-related. With more money than any individual claimant, defendants would probably be able to marshal more expertise and put it to better use.[12]

Nevertheless, scientific studies can also be of considerable value to individual claimants, and several occupational health advocates have recommended various means for structuring their responsible use. One proposal suggests using expert panels to assist boards in evaluating evidence.[13] Another recommends establishing "presumptive standards" that would presume a plaintiff was eligible for compensation, if sufficient epidemiological and toxicological evidence supported such a finding, and the defendant was unable to rebut the presumption.[14]

These issues may not be of great importance at present in terms of claims for reproductive harms because of the paucity of compensation claims in this area, as well as the difficulty in detecting reproductive problems in populations of workers. Nevertheless, these factors can be expected to increase in importance over time as knowledge increases about workplace exposures and their reproductive implications. Only the claims that survive the obstacles discussed earlier ("personal" injury and, in most States, disability) will ultimately face the causation test (see table 9-3).

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[10] Solomons, Workers' Compensation for Occupational Disease Victims, Workmen's Comp. L. Rep. (CCH) 11 (1977).

[11] M. Baram, supra note 1.

[12] E. Tanenhaus, Administration, Coordination and Trial of Workers' Compensation Occupational Disease Claims, in Occupational Disease Litigation (S. Birnbaum (ed.) (1983)).

[13] F. Goldsmith, Occupational Safety and Health 193 (1982).

[14] American Public Health Association, Policy Statement No. 8329 (PP): Compensation for and Prevention of Occupational Disease, vol. 74, No. 3, Am. J. Pub. Health 292, 294 (March 1984).

In the majority of States that require disability, the surviving claims would be those for serious and incapacitating injuries to reproductive organs, pregnancy-related illness, and miscarriage; in the minority of States that permit compensation for nondisabling loss of function, the surviving claims might also include sexual dysfunction, infertility/sterility, early menopause, and breast milk contamination. Those surviving claims that are for reproductive harms to workers arising from workplace accidents or physical stresses generally will not raise new or especially difficult causation issues, but those for reproductive diseases suffered by the worker may involve substantial evidentiary problems of causation.

Other Requirements

State laws also impose a number of other conditions on eligibility for compensation. For example, the worker must be one who is not exempt from workers' compensation coverage under the law (e.g., in some States, agricultural, domestic, and other workers may be exempt), and who also has employee status (rather than independent contractor status) under the law.

In most States, an injury or disease that had preemployment or extra-employment sources may be compensable if evidence establishes that it was accelerated or aggravated by employment circumstances. Several States still require that a disease, to be compensable, must have been specified as compensable in the basic statute. As has been noted, although most States require the disease to be one that arises "out of and in the course of employment," some States also require that the

Table 9-3.—Summary of Harms, Victims, Benefits Criteria, and Causation Problems

	Victim		
Circumstances of harm	Worker	Spouse	Fetus and offspring
1. Accidental injury to worker reproductive system or fetus	Personal injury: eligible for compensation for medical benefits in all States and loss of function and disfigurement in a few States. No disability benefits unless earnings loss. No special causation problems.	No personal injury, therefore, no compensation	No personal injury, therefore, no compensation
2. Physical stress on worker	Personal injury: eligible for compensation for medical benefits in all States and loss of function in a few States. No disability benefits unless earnings loss. No special causation problems.	No personal injury, therefore, no compensation	No personal injury, therefore, no compensation
3. Acute or chronic exposure of worker, spouse, or fetus	If personal injury, will be eligible for compensation for medical benefits in all States and loss of function benefits in a few States. No disability benefits unless earnings loss. Special causation problems.	No personal injury, therefore, no compensation	No personal injury, therefore, no compensation
4. "Side effect" cases where reproductive function impaired due to other injuries or diseases	Probably not applicable, since other injury or disease will be primary personal injury for disability compensation, not the reproductive injury.	NA	NA

NA—Not applicable.

SOURCE: Office of Technology Assessment.

harm be peculiar or unique to employment. States with this narrow view may refuse to compensate so-called ordinary diseases of life that may be contracted outside of the workplace.

Reproductive Harm Claims Experience

Numerous studies and data collection activities have focused on claims under the various workers' compensation systems, and occupational disease claims in general. No study has yet focused on claims involving reproductive harms. Moreover, the occupational disease claim studies do not contain categories or separate entries for harms, claims, board decisions, or settlements related to reproductive functions. Since these disease studies have been conducted by various insurers and insurance associations, employers and trade associations, academicians, Federal and State governments, and other interested organizations, the dearth of data or interest in reproductive claims can probably be attributed, at least in part, to the low incidence of such claims, and their consequent lack of economic or social significance to those conducting the studies.

Because of this lack of available data on reproductive claims, OTA contacted the State compensation boards for each of the 50 States seeking information on coverage of reproductive injuries and diseases that were job-related, and asking for citations or references to any relevant decisions or studies. No responses offered references to cases or studies. Two State boards (Florida, Minnesota) mentioned that anatomical injuries to male sex organs had led to several claims. The other States provided no information as to the incidence of reproduction-related workers' compensation claims or types of injuries or diseases. One State board (Kentucky) had no recollection of any such claims during the last 12 years, and another (Kansas) observed that State statistical studies do not provide the information sought; this condition probably prevails in most States.

A review of the reported legal cases yielded a small collection of workers' compensation cases involving reproductive harms. The actual incidence and types of claims for reproductive harms could not be assessed, however, because claim files are sealed, and board decisions and settlement outcomes are unpublished in virtually all States. Further, although researcher access to claims files may be provided if provision is made for claimant privacy rights, most States do not organize or label their thousands of files by types of claims (e.g., disease, injury). In addition, it appears to be common practice for insurers as defendants to settle most disease claims, including reproductive damage claims, and avoid the costs and risks of full hearings. The costs arising from such settlements can be recaptured by the insurer over the next few years by means of adjusting the cost of insurance to the employer. **It appears that the best possible source of claims information—the records of workers' compensation insurers—is unavailable to most researchers and therefore remains unused for purposes of public policy analysis.**

The incomplete picture that emerges indicates that historically the most common uses of the workers' compensation systems for redress of reproductive harms involve accidental injuries to male workers, primarily injuries to male genitalia and injuries that lead to male impotence for either physical or psychological reasons.

Only a few of the tens of thousands of workers' compensation claims that have been appealed to State courts have involved reproductive harm claims due to chronic exposure to chemical, physical, and biological agents. Such claims may increase as recognition grows of the reproductive and developmental effects of these agents, but compensation would be limited to the worker, not the spouse or offspring, under the personal injury criterion.

Exclusivity of Remedy

Because only a few of the many types of reproductive harms are "personal" and therefore subject to workers' compensation law, and still fewer are compensable because of a lack of job disability and because amounts of compensation, if any, will be low in most cases, workers and their families increasingly seek common law remedies. By suing the employer or other party, under any of the several theories of liability at common law, a worker or a member of his or her family may be able to secure more ample remedies in the form of compensatory and punitive damages.

But the "exclusivity of remedy" doctrine embedded in State compensation statutes has traditionally been construed by State courts as barring tort actions by the worker against the employer, even if the worker does not file a worker's compensation claim. Remedies for nonpersonal injuries (those to the worker's spouse, fetus, offspring, and descendants) are not disturbed by the exclusivity doctrine because they are not covered under State compensation law.

The exclusivity doctrine has withstood worker challenges as an unconstitutional denial of due process. It has instead been viewed by the courts as part of a system that constitutes a rational exchange by which employees, in theory, are guaranteed swift disposition of claims and provision of monetary payments. Over the years, two narrow exceptions to the exclusivity doctrine have evolved (the intentional tort and dual capacity exceptions). These are discussed in chapter 10.

The bar to worker tort suits against employers and their insurers has generally been maintained by the courts without regard to whether the worker's claim actually resulted in the payment of benefits. One of the early (1921) leading cases involved a personal injury to the claimant's pubic nerve, arising from an accident on the job, which resulted in sexual impotence for which no job disability was shown. The claimant, denied disability benefits, sought to sue the employer in tort. The court refused to permit the common law action on the basis of the compensation statute's exclusivity provision, and concluded that any changes in the law to provide relief in such cases of job-related injuries that did not impair wage-earning capacity should come about by legislative, not judicial, action.[15]

Inequitable outcomes in which the claimant is denied any compensation under *both* the workers' compensation and common law systems have led a few State legislatures to enact "loss of function" categories of benefits. But in the absence of such remedial legislative amendments, the problem has been left to the courts.

The harshness of the exclusivity rule has led some courts to provide workers' compensation for functional or health impairments without job disability. A 1952 case[16] concerned a male worker who had been exposed to airborne particles of female hormones, allegedly resulting in breast development and impotency. The worker filed suit, claiming that the workers' compensation statute did not apply because he did not suffer an occupational disease under the State compensation law. The court disagreed, but held that a permanent injury involving the loss of a physical function used in the ordinary pursuits of life was compensable under the compensation statute even if there were no disability or wage loss. Such interpretations of statutory language on occupational disease may become more widespread if State legislatures fail to respond.[17]

Nonetheless, **most courts steadfastly maintain the exclusivity doctrine, and bar tort actions by workers against their employers without considering the worker's inability to secure the statutory remedy.** Thus, in a recent personal injury suit by a worker and his wife seeking common law damages from his employer for sterility claimed to be caused by workplace exposure to the chemical DBCP, the Michigan Court of Appeals held that the State workers' compensation act's exclusivity provision barred the suit, even though the worker's sterility was not compensable under the compensation act. A dissenting opinion argued for a more humane judicial approach:

> [t]he right to procreate is basic. Procreation constitutes a fundamental human experience. The Legislature could not possibly have intended to include deprivation of an employee's ability to procreate, accomplished in the insidious manner alleged in this case, as a personal injury or disease subject to the Worker's Disability Compensation Act. "Personal injury" and "disability" as used in the Act connote inability to perform labor, not inability to procreate. Sterility in and of itself is not compensable under the Act. . . . Plaintiffs should have their day in court.[18]

Other recent decisions involving alleged reproductive harms from chemical exposure have also maintained the barrier to tort remedies. The most recent involved five workers who brought a tort action against their employer, claiming that their exposure to DBCP resulted in carcinogenesis, mutagenesis, and sterility. The court dismissed the tort action on the ground that the claims were subject to the exclusive jurisdiction of the State workers' compensation statute:[19]

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[15] Hyett v. Northwestern Hospital, 147 Minn. 413, 80 N.W. 552 (1921).

[16] Stepnowski v. Specific Pharmaceuticals, 18 N.J. Super. 495, 87 A.2d 546 (1952).

[17] See generally P. Barth, Workers' Compensation and Work-Related Illnesses and Diseases 26 (1980).

[18] Cole v. Dow Chemical Co., 112 Mich. App. 198, 315 N.W.2d 565 (1982).

> [i]t is true that neither sterility, carcinogenicity, nor mutagenicity are scheduled injuries, unless one were to construe them as constituting partial loss of use of testicles. . . . Nor are they disabling conditions in themselves. Nonetheless, this does not mean that plaintiffs have no remedies under the Workers' Compensation Act. Claims based on psychological employment disabilities are compensable under the Act. [Citations omitted.] It is clear that the allegations of the complaint, if taken as true, would bring plaintiffs within the scope of the Act, and that under the Act, plaintiffs would be entitled to be considered for some form of relief.[20]

The court concluded that:

> [b]ased on the allegations in the instant action, it is possible that plaintiffs would be entitled to medical expenses. . . . Any work-related physical or psychological earning disabilities would possibly be compensable. . . . The inadequacy of the award or the complete lack of an award, under the Workers' Compensation Act, cannot furnish the basis of a common law cause of action. So long as the accidental injury, occupational disease or infection arises out of and in the course of the employment, the Workers' Compensation Act affords the exclusive remedy.[21]

Nevertheless, decisions involving a variety of other types of injuries indicate that the exclusivity doctrine has been eroding, and tort actions increasing, for several reasons. Courts in several States now permit workers to sue employers irrespective of whether the worker's job-related injury is statutorily compensable. In these cases, the courts in some States have refused to permit the exclusivity rule to protect the employer from tort liability when the employer acted negligently,[22] acted in a "dual capacity" (e.g., as both employer and manufacturer of the product that harmed the employee),[23] or acted in a willful, deliberate, or intentional manner to cause the worker's injury.[24] These exceptions are discussed in greater detail in chapter 10.

Because of these judicial decisions, the exclusivity doctrine is now at a crossroads, with strong pressures being exerted on legislatures to enact liberalizing reforms due to concerns about fairness. **In the absence of Federal legislation, each State will continue to grapple with the boundaries of the exclusivity doctrine and how to deal fairly with reproductive harms to workers. If an increase in reproductive harms occurs, and causal linkages to workplace exposure become clearer, the problem of workers and other parties adversely affected who either have no remedies or, at most, inadequate remedies in the worker compensation system will become more acute.** These potential parties will press forward with common law actions of various types, discussed in chapter 10.

Conclusion

Most workers who are reproductively harmed are not entitled to workers' compensation, despite the fact that State workers' compensation statutes are designed to provide compensation for injuries and diseases that occur in the course of employment. In addition, an employee covered by a workers' compensation statute generally cannot sue his or her employer for any injury or disease subject to the statute.

The three major requirements that are common to most if not all State compensation systems that affect a worker's ability to secure benefits for reproductive harms caused by workplace exposures are: 1) the requirement of a *"personal" injury or*

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[19] Vann v. Dow Chemical Co., 561 F. Supp. 141 (W.D. Ark. 1983).

[20] Id. at 144-45.

[21] Id. at 145.

[22] See, e.g., Ferriter v. D. O'Connell's Sons, 381 Mass. 507, 413 N.E.2d 690 (1980); Reed Tool Co. v. Copelin, 610 S.W.2d 736 (Tex. 1981).

[23] See, e.g., Bell v. Industrial Vangas, Inc., 30 Cal. 3d 268, 637 P.2d 266, 179 Cal. Rptr. 30 (1981); Douglas v. Gallo Winery, 69 Cal. App. 3d 103, 137 Cal. Rptr. 797 (1977). For U.S. Supreme Court recognition of dual capacity under a Federal compensation program, see Reed v. The Yaka, 373 U.S. 410 (1963).

[24] See, e.g., Johns Manville Corp. v. Contra Costa Superior Court, 27 Cal. 3d 465, 612 P.2d 948, 165 Cal. Rptr. 858 (1980); Blankenship v. Cincinnati Milacron Chemicals, Inc., 69 Ohio St. 2d 608, 433 N.E.2d 572 (1982); Mandolidis v. Elkins Industries, Inc., 246 S.E.2d 907 (W. Va. 1978).

disease, 2) the requirement that the injury or disease result in *job disability*, and 3) the requirement that the injury or disease be *caused* by a workplace accident or exposure.

The requirement of a "personal" injury or disease precludes compensation for injuries or diseases suffered by others, such as the worker's spouse, fetus, child, or descendant. Thus, if the condition is job-related and impairs the male worker's ability to cause conception (e.g., by causing impotence, infertility, sterility) or the female worker's ability to conceive and carry a fetus to term (e.g., infertility, sterility, spontaneous abortion, miscarriage), the disease or injury is considered personal to the worker and is eligible for compensation so long as it meets various other criteria. Conversely, if the condition is one that has not prevented conception or birth, but instead impairs the worker's fetus, child, spouse, or descendants, the doctrine of personal injury or disease as a condition for securing workers' compensation would prevent financial recovery. In most States, the personal injury criterion precludes claims for reproductive harms that involve the developing offspring, including birth defects, decreased birthweight, change in gestational age at delivery, altered sex ratio, multiple births, infant death, and childhood morbidity or mortality.

A reproductively harmed worker can generally recover medical benefits for medical expenses incurred if his or her medical problem meets the personal injury criterion and the worker can prove the job relatedness of the injury. A worker who loses sexual or reproductive function may want additional benefits to compensate for the lost function, but unless the worker is disabled, he or she will often be unable to collect a monetary substitute under the workers' compensation system. The requirement of disability prevents the award of nonmedical benefits for most claims of reproductive injury or disease, since such harms do not usually disable the worker or prevent him or her from resuming work at the same job. Of the few reproductive endpoints that meet the personal injury criterion discussed above, only injury to reproductive organs, illness during pregnancy, and fetal loss are likely to result in any temporary job disability. When a reproductive harm is sufficiently disabling to prevent the employee from performing the job for a temporary or permanent period, as in the case of a job-induced miscarriage, the worker is entitled to collect disability benefits. However, because of the short duration of the period of actual disability, such workers are probably more likely to take advantage of employer-provided sick leave benefits than face the expense, risk of the claim being denied, loss of medical privacy, and low benefits endemic to workers' compensation claims. Thus, although disability compensation is theoretically available to a small number of reproductively harmed workers, they are unlikely to claim this entitlement.

Causation evidence is required in each State's compensation system, because the governing statutes typically require that compensation coverage and benefits apply only to claims arising out of and in the course of employment. Usually, the claimant has the burden and expense of proving by a preponderance of the evidence that the injury or disease is job-related. Proving causation is complicated by the fact that compensation board attitudes toward the admissibility and weight to be accorded scientific evidence, particularly toxicological or epidemiological evidence of a probabilistic nature, have been cautious, skeptical, and inconsistent. Boards generally still prefer medical evidence that a particular individual contracted a particular disease in a particular way, to scientific evidence that shows how many, or even most, people contract the disease. Both workers claiming benefits for occupational disease and insurers defending against such claims are unhappy with this situation and believe that a more flexible approach by boards and courts would work to the advantage of their differing interests. The causation problem is endemic to disease claims in general.

Because only a few of the many types of reproductive harms are compensable under the workers' compensation system, workers increasingly seek common law remedies. But the "exclusivity of remedy" doctrine embedded in most workers' compensation statutes provides that an employee covered by a workers' compensation statute cannot sue his or her employer for any injury or disease subject to the statute. This bar to worker suits has generally been maintained by the courts *without regard to whether the worker's claim actually resulted in the payment of benefits.* This is especially troublesome in the case of job-induced reproductive harms because the workers' compensation system usually fails to award benefits for reproductive problems, yet employees with job-related reproductive problems are precluded from suing their employers. The harshness of the exclusivity rule has led some courts to provide compensation for functional or health impairment without job disability. Other courts have expanded the list of exceptions to the rule for cases of dual capacity and intentional torts. Nonetheless, most courts steadfastly maintain the exclusivity doctrine and bar actions by employees who claim they have occupationally induced reproductive harms. This has generated concerns about the fairness of the compensation system.

If an increase in reproductive harms occurs, and causal linkages to workplace exposure become clearer, the problem of workers and other parties adversely affected who either have no remedies or, at most, inadequate remedies in the workers' compensation system will become more acute. These victims of hazardous occupational exposures will by default bear the burden of their occupational exposures to reproductive health hazards.

Chapter 10

Tort Liability for Reproductive Harm

CONTENTS

Page
Introduction to the Common Law of Torts 301

Types of Injuries and Potential Plaintiffs 301

Theories of Liability 302
  Negligence 302
  Strict Liability 304
  Product Liability 304
  State-of-the-Art Defense 307
  Fraud 308
  Breach of Warranty 309
  Prenatal Torts 309
  Viability 310
  Pre-conception Torts 312
  Wrongful Life 312

Intangible Injuries Resulting From Reproductive Health Hazards 313
  Loss of Consortium 313
  Emotional Distress 315

Suits Against Employers: The Exclusivity Rule, Revisited 316
  Dual Capacity Exception 316
  Intentional Tort Exception 318

Defendants 319
  Potential Defendants 319
  Deceit 319
  Multiple Defendants 319
  Liability When Defendant Is Unidentifiable 320
  The Problem of Bankruptcies and Successor Corporations 321

Legal Causation 322
  Proving Legal Causation 322

Other Considerations 324
  Statutes of Limitations and Repose 324
  Prior Litigation 324
  Sovereign Immunity 325
  Conclusion 326

Figure

Figure No. *Page*
10-1. Average Expenditures per Asbestos Product Liability Claim, Jan. 1, 1980-Aug. 26, 1982 305

Tort Liability for Reproductive Harm

INTRODUCTION TO THE COMMON LAW OF TORTS

The common law, as distinct from statutory law, comprises the body of rules and principles used by courts in the absence of applicable legislation. It derives its authority solely from the judgments and decrees of courts applicable to persons, property, and government. Legislation may either modify or codify the common law.

A tort is a civil wrong, other than breach of contract, for which the common law provides a remedy. Although the common law in most States has common roots and has usually developed along similar lines, there is more diversity among States in the law of torts than in most other areas of the law. Perhaps more than any other branch of the law, tort law is a battleground of social theory. Its primary purpose is to make a fair adjustment of the conflicting claims of the litigating parties. But the 20th century has brought increasing realization of the fact that the interests of society in general may be involved in private disputes.[1]

Workers' compensation statutes represent one form of legislatively mandated modification to State common law. However, as discussed in chapter 9, workers' compensation laws as they currently exist frequently offer little or no compensation for job-induced reproductive failure or harm. As a result, workers and their families may resort to tort litigation in increasing numbers, to the extent that this is not barred by the exclusivity of remedy doctrine (discussed in chapter 9).

Employees and their families presently have narrow opportunities to bring common law actions for personal injuries against employers, and the employer's hired physicians and other health professionals. But these opportunities vary from State to State, and do not yet amount in *any* State to a comprehensive and consistent social policy for imposing or refusing to impose liability for reproductive injuries to employees and their families, beyond that available under workers' compensation statutes. Employees have therefore sought easier pathways for securing compensation and punitive damages. The primary pathway involves litigation against a third party: generally, another firm that furnished to the employer "defectively dangerous" products or negligently performed services. **Product liability theory, at present, affords employees and their families their best opportunity to obtain substantial damage verdicts.**

This chapter explores the opportunities for and barriers to securing common law tort remedies.[2]

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[1] See W. Prosser, The Law of Torts (4th ed. 1971).

[2] It should be noted that many of the decisions discussed in this chapter are lower court decisions. Lower court decisions are generally limited in application and authority, and may be reversed on appeal.

TYPES OF INJURIES AND POTENTIAL PLAINTIFFS[3]

Various types of injuries to reproductive health can arise from a worker's exposure to occupational hazards. These injuries can be classified in many ways. For example, they can occur at three different times: before conception, during pregnancy, or after birth.

Injuries that occur *prior to conception* may harm the reproductive health of the male or female worker, the worker's spouse, or both. Some of these impairments may be identifiable before conception (e.g., sterility, impotency, sperm and ova abnormalities, sexual dysfunction) and may prevent or diminish the possibility of conception, impair maternal adaptation to pregnancy, or lead to a conception that later results in an adverse outcome. However, some pre-conception injuries, such as chromosomal mutations in the ovum or sperm, may not be identified until manifested in adverse outcomes such as fetal loss, birth defects, chromosomal abnormalities in offspring, or genetically caused disabilities and susceptibilities. Pre-conception injury may also lead to other problems, including emotional distress for the worker, spouse, and offspring, loss of sexual and emotional companionship (consortium) for the worker and spouse, and even loss of parental companionship and resources for other children. Pre-conception injury may possibly result in adverse effects in future generations.

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[3] For purposes of this report, the word "injury" is defined as any damage to health caused by impact, exposure, disease, or other means.

Reproductive injuries that occur *during pregnancy* may endanger the health of the fetus or complicate the pregnancy and endanger the health of the pregnant woman. These injuries may affect the fetus either before or after it is able to live outside the uterus, and may or may not result in fetal loss. Like pre-conception injuries, these injuries may also result in emotional distress and loss of sexual and parental companionship, thereby resulting in harm to the pregnant worker's husband and any other children she may have.

Postnatal injuries within the context of the reproductive cycle are those which may harm the infant through exposure to an exposed parent, as where a parent brings home hazardous fibers on his or her clothing, or the mother's breast milk is contaminated by her exposure to a hazardous chemical. In addition to any physical injuries, such exposure may also result in emotional distress for both parents and child.

The parties who may suffer these reproductive harms include the:

- male or female worker;
- worker's spouse and children in being;
- embryo, fetus, or infant (depending on when the injury occurred and whether the conceptus survived); and
- the descendants.

THEORIES OF LIABILITY

Negligence

Negligence is the failure to use such care as a reasonably prudent and careful person would use in similar circumstances. However, liability for negligence requires more than mere conduct. The traditional formula for the elements necessary to prevail in a negligence suit may be stated as follows:[4]

- A *duty,* or obligation, recognized by the law, requiring the actor to conform to a certain standard of conduct for the protection of others against unreasonable risks.
- A *breach of duty,* or failure to conform to the standard required. The failure to conform may result either from inaction when action is legally required, or action which fails to conform to the legal standard.
- A reasonably close *causal connection* between the conduct and the resulting injury. This is commonly known as "legal cause" or "proximate cause."
- *Actual loss, injury, or damage* to the interests of another. Nominal damages to vindicate a technical right cannot be recovered in a negligence action where no actual loss has occurred. The threat of future harm, not yet realized, is not generally considered to be an actual loss for which recovery may be granted. Some recent cases have, however, found an actual injury to exist when a plaintiff fears for his or her future health due to the defendant's negligent act. The actual damage is not the possible future harm itself, but the emotional anguish created by the plaintiff's knowledge of exposure and likely future effects.[5]

Duty and Breach of Duty: The Reasonable Person Standard

The theory of negligence presupposes a uniform standard of behavior to which one has a duty to conform. Yet the infinite variety of situations that may arise makes it impossible to fix definite rules in advance for all possible human conduct. The most that can be done is to devise a formula that can be applied by courts and juries.[6]

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[4] W. Prosser, supra note 1, § 30 at 143; Restatement (Second) of Torts § 281 (1965).

[5] Nat'l. L.J., May 28, 1984, at 1, col. 1.

The courts have dealt with the difficult problem of creating a standard that can apply to all people in all situations by "creating a fictitious person . . . the 'reasonable man of ordinary prudence.' "[7]

The reasonable person standard of conduct is a personification of a community ideal of reasonable behavior. Members of the community—including workers and employers—are required to act with *due care,* that is, as the hypothetical reasonable person would act in identical circumstances. Failure to conform to the reasonable person standard of conduct imposed by negligence law may result in liability if the causation and loss requirements are met.[8]

Negligence is conduct that falls below this standard for the protection of others against unreasonable risk of harm. The legal concept of risk necessarily involves a recognizable danger, based on some knowledge of existing facts, and some reasonable belief that harm may follow. In its legal use, a risk is a danger which is, or should be, apparent to the reasonable person. (The legal definition of "risk" is essentially an amalgam of the scientific definitions of "risk and "hazard," as discussed in chapter 2.) In light of the recognizable risk, one must act reasonably, and the defendant's honest blunder or mistaken belief that no harm will result will not legally excuse his or her conduct (though it may morally excuse it) if a reasonable person exercising due care would not have so acted. Nearly all human acts carry some recognizable but remote possibility of harm to another, but these are not unreasonable risks. Conversely, if the risk is an appreciable one, and the possible consequences are serious, the question is not one of mathematical probability alone:

> The odds may be a thousand to one that no train will arrive at the very moment that an automobile is crossing a railway track, but the risk of death is nevertheless sufficiently serious to require the driver to look for the train.[9]

Generally, as the gravity of the possible harm increases, the apparent likelihood of its occurrence need be correspondingly less for a legal duty to attach. This is so because a reasonable person would consider these circumstances in deciding on a course of action.

Negligence is a fault-based standard since liability is imposed only on a party whose fault (i.e., failure to act as a "reasonable person") led to the injury. The concept of imposing liability for harms on those who were at fault in causing those harms has considerable appeal. In practice, however, it is not always a simple matter to demonstrate that specific conduct gave rise to exposure to a reproductive health hazard.[10] Moreover, it has been observed that plaintiffs in negligence suits involving toxic exposures may have difficulty in establishing that the defendant was at fault in causing their exposure.[11] Often no regulatory, industry custom, or common sense exposure standards apply to the substance in question. In the absence of such standards, plaintiffs are forced to produce evidence on the risks known or theoretically knowable at the time of exposure (and the costs of discovering unknown but knowable risks), as well as on the means of controlling those risks, in order to establish what standard of conduct should have been followed in the circumstances.

Thus, negligence may occur in a multitude of contexts in which reproductive risks are generated, including the:

- design, operation, maintenance, or monitoring of workplaces where reproductive health hazards are present;
- design, testing, construction, inspection, quality control, or labeling of products posing reproductive risks, or the provision of warnings or instructions for their safe use;
- provision of medical or other expert services to persons encountering reproductive health hazards;

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[6] W. Prosser, supra note 1, § 32 at 149-50.

[7] Id.

[8] For a comprehensive discussion of the law of negligence, see W. Prosser, supra note 1.

[9] W. Prosser, supra note 1, § 31 at 147.

[10] See generally Ginsberg & Weiss, Common Law Liability for Toxic Torts: A Phantom Remedy, 9 Hofstra L. Rev. 859, 887 (1981).

[11] See, e.g., Trauberman, Statutory Reform of "Toxic Torts": Relieving Legal, Scientific and Economic Burdens on the Chemical Victim, 7 Harv. Envtl. L. Rev. 177, 192-97 (1983).

- conduct of independent or regulatory inspections of sites where reproductive health hazards are present; and the
- legal or collective bargaining representation of the interest of persons exposed to reproductive health hazards.

Strict Liability

The legal doctrine of strict liability for abnormally dangerous activities imposes liability for harm caused as the result of certain unusually risk-laden activities, regardless of whether the defendant was negligent in failing to avoid the injuries.[12] The basis for creating liability in the absence of fault was first enunciated over a hundred years ago in a landmark British case:

> We think that the true rule of law is that the person who for his own purposes brings on his land and collects and keeps there anything likely to do mischief if it escapes, must keep it at his peril, and . . . is . . . answerable for all the damage which is the natural consequence of its escape.[13]

In this country, the activities to which the strict liability rule has been applied include storage of explosives or flammable liquids, blasting, pile-driving, crop-dusting, and fumigation of a part of a building with cyanide gas.[14] The American Law Institute's *Restatement (Second) of Torts* provides the following guidelines for determining what activities might be abnormally dangerous within the meaning of this rule:

a. existence of a *high degree of risk* of some harm to the person, land, or chattels of others;
b. likelihood that the *harm* that results from it will be great;
c. *inability to eliminate the risk* by the exercise of reasonable care;
d. extent to which the activity is *not* a matter of *common usage;*
e. *inappropriateness* of the activity to the place where it is carried on; and
f. extent to which the activity's *value to the community* is outweighed by its dangerous attributes.[15]

No reported judicial decision has yet considered whether an activity should be deemed abnormally dangerous because it creates a reproductive health risk. Indeed, nothing in the rule of strict liability necessarily compels the conclusion that either the generation, storage, transportation, handling, or use of materials posing reproductive health hazards is necessarily abnormally dangerous for the purposes of imposing liability without fault. In most jurisdictions, the determination of whether an activity is abnormally dangerous is made on a case-by-case basis. Application of the doctrine of strict liability is not automatic, even for a class of activities with similar risks, and will depend on a factual finding that the particular activity at issue is abnormally dangerous.[16]

The doctrine of strict liability offers an opportunity for those who experience reproductive harms to recover from those engaged in activities causing those harms even in the absence of negligence. The availability of strict liability, however, is substantially restricted by the requirement that the activity in question be abnormally dangerous. As is the case with negligence, the proof required on this issue can be quite complex and technical. Moreover, the factors enumerated in the *Restatement* could well result in a finding that the activity at issue was not abnormally dangerous. In such a case, ordinary care would be used as the basis for imposing liability.

Product Liability

Product liability law is composed of the set of principles that govern a product seller's responsibility for harms caused by its products. The law allows persons who are injured because of exposure to a "defective" and "dangerous" product to seek compensation for their injuries from anyone who participated in placing the product into the stream of commerce, including the manufacturer, wholesalers, and retailers. In most States, such parties will be liable, regardless of fault or negligence, if their product is found to be in a "defective condition" that makes the product "unreasonably dangerous" to the user or consumer.[17]

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[12] Restatement (Second) of Torts § 519 (1965).

[13] Fletcher v. Rylands, 3 H. & C. 774, 159 Eng. Rep. 737 (1865), rev'd. 1 L.R. Ex. 265 1866, aff'd., 3 L.R. H.L. 330 (1868).

[14] See W. Prosser, supra note 1, § 78 at 509-10.

[15] Restatement (Second) of Torts § 520 comment g (1965).

[16] But see New Jersey Dept. of Environ. Protect. v. Ventron, 94 N.J. 254, 463 A.2d 893 (1983) (disposal of toxic wastes ruled to be abnormally dangerous under all circumstances).

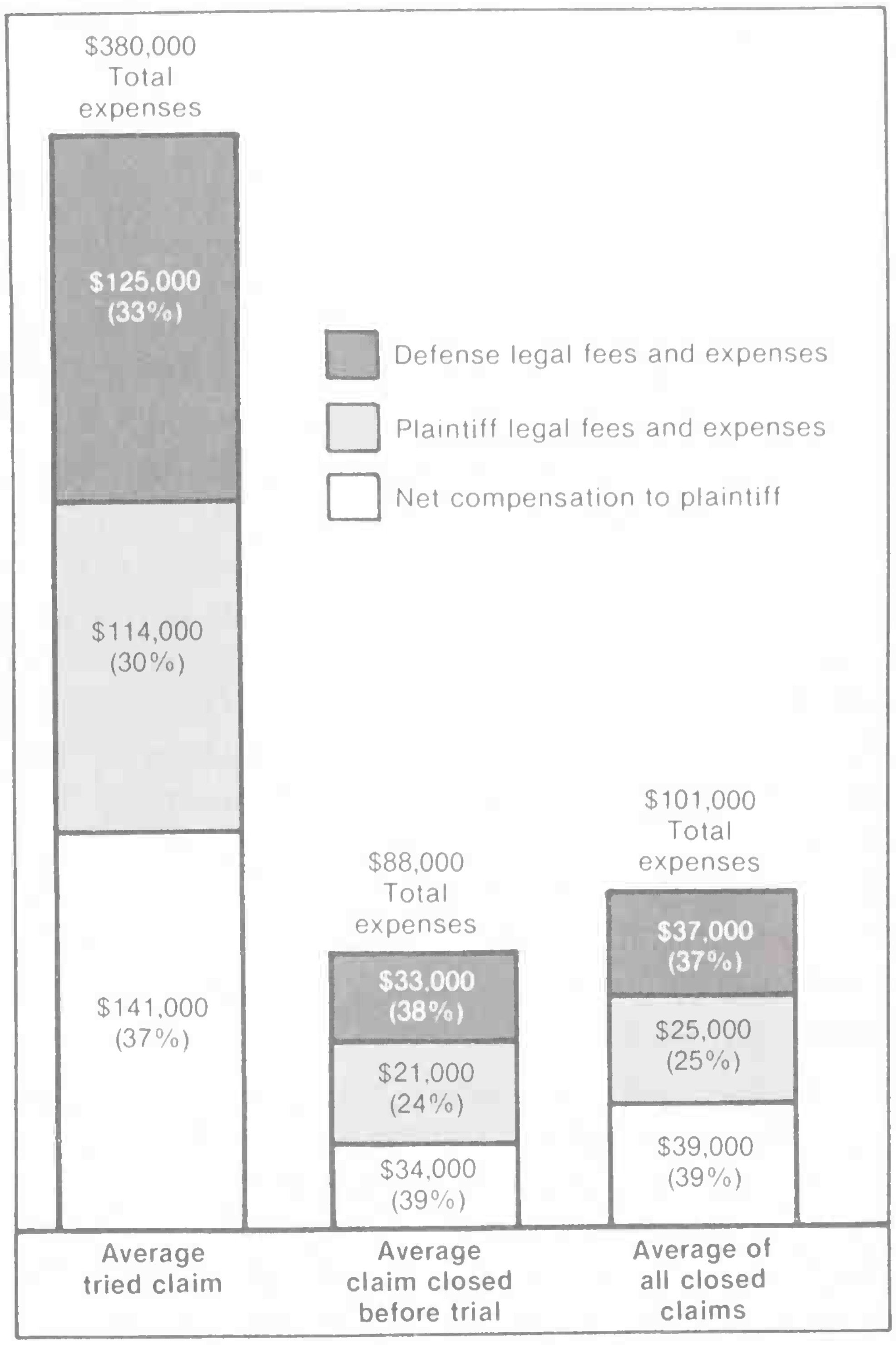
This liability extends not only to injured purchasers and users, but to bystanders (co-workers) and other third parties as well.[18] A defect may be in either the design or manufacture of the product, or in the failure to adequately communicate product hazards or safe use instructions.

The last two decades have seen a sharp increase in product liability lawsuits involving toxic substances. The plaintiffs in these suits allege that they were exposed to products containing toxic substances; that these products were defective in design, manufacture, or labeling; and that these defects caused a disease.

While no data have been collected concerning the costs of product liability litigation for diseases caused by reproductive health hazards, data concerning product liability claims for diseases caused by asbestos exposure are instructive. (See figure 10-1.) According to a study by the Rand Corp., in the average asbestos lawsuit that actually went to trial, the plaintiff's net award was $141,000. In addition, plaintiff and defendant spent a total of $239,000 on legal fees and the various expenses associated with a trial (e.g., witness fees, investigator's report, consultations with experts). In the average asbestos lawsuit that was settled before trial, the plaintiff's net compensation was $34,000, while the parties' legal expenses were $54,000. Since the vast majority of personal injury lawsuits are settled prior to trial, it is not surprising that the average asbestos claim approached the nontrial figures: plaintiff's net compensation totaled $39,000 and legal expenses totaled $62,000 for both sides. These figures do not include the costs borne by Federal and State governments for court administration.[19]

A National Council on Compensation Insurance (NCCI) report provides some information about workers' compensation claims for asbestosis.[20] NCCI found that the average asbestosis claimant in the workers' compensation system received $25,800. From the data, it appears that this sometimes includes plaintiff's legal fees.

Figure 10-1.—Average Expenditures per Asbestos Product Liability Claim, Jan. 1, 1980-Aug. 26, 1982



SOURCE: Rand Corp. adapted by *ABA Journal* (1984).

Although not directly comparable with the Rand Corp.'s data for various reasons,[21] the NCCI data provide a basis for cautious comparison of the tort and workers' compensation systems. A cautious comparison of compensation for asbestos-related diseases tends to support the preference of plaintiff's lawyers for filing tort suits rather than workers' compensation claims when the legal criteria for product liability is met.

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[17] Restatement (Second) of Torts § 402A (1965).

[18] Sills v. Massey-Ferguson, Inc., 296 F. Supp. 776 (D. Ind. 1969); Elmore v. American Motors Corp., 70 Cal. 2d 578, 451 P.2d 84, 75 Cal. Rptr. 652 (1969).

[19] Hearings on the Occupational Disease Compensation Act of 1983: Hearings on H.R. 3175 Before the Subcommittee on Labor Standards of the House Committee on Education and Labor, 98th Cong., 1st sess. 310 (1983) (Report on the Costs of Asbestos Litigation by the Rand Corp.).

[20] National Council on Compensation Insurance, Workers' Compensation Claim Characteristics 1984.

[21] For example, the NCCI information reported here concerns only the most prevalent asbestos-produced disease, asbestosis, while the Rand information reflects all asbestos-related diseases. In addition, the NCCI surveyed workers' compensation insurers alone, and not companies that self-insure. While it is not clear that these distinctions are relevant, the data should nevertheless be interpreted with caution.

It is interesting to note that, notwithstanding the workers' compensation system's goal of providing swift compensation, NCCI found that as of 18 months after workers reported having the disease, 51 percent of the asbestosis claims were still open and unresolved.[22]

A person who suffers a reproductive injury cannot bring a product liability suit merely by showing that his or her harm arose out of the use of a product. Rather, it is necessary to demonstrate that the product contained some character that is both a defective condition and unreasonably dangerous.[23] The prevailing interpretation of "defective" is that the product does not meet the reasonable expectations of the ordinary consumer as to its safety. It has been said that this amounts to saying that if the seller knew of the product's condition, he or she would be negligent in marketing the product.[24]

A "defect" may take several forms. The conceptually simplest is the *manufacturing defect*. Such a defect results from a mistake in the manufacturing process, in quality control, or in the handling of the product prior to its sale. The basic allegation of a manufacturing defect case is that "something went wrong" during the manufacturing or handling process that caused the product to fall below the standard for the product line. A typical manufacturing defect action alleges that the product failed to conform to the manufacturer's own specifications. For example, a chemical that has been contaminated with a foreign substance would be defective (though not necessarily unreasonably dangerous). Typically, a manufacturing defect will appear in only a small number of units of a product and is identifiable by its differences either from otherwise identical units of the same product or from the manufacturer's specifications, warranties, or performance standards. In such cases, it is not necessary to produce any evidence as to how the defect arose, how it went undiscovered, or even whether the manufacturer *could* have discovered the defect. The defendant's fault or negligence is not an issue.

In contrast, a *design defect* is much more difficult to define in product liability cases. In design defect cases, the products do meet the manufacturer's specifications and standards, and the alleged defect arises from a mistake in the formulation or conceptualization of the product. The allegation in a design defect case is either that the manufacturer should have formulated the product differently or that the product never should have been marketed at all.

The relevant factors to consider in evaluating whether a product is defective in design include:

- any warnings or instructions provided with the product;
- the technological and practical feasibility of a product designed and manufactured so as to have prevented harm while substantially serving the likely user's expected needs;
- the effect of any proposed alternative design on the usefulness of the product;
- the comparative costs of producing, distributing, selling, using, and maintaining the product as designed and as alternatively designed; and
- the new or additional harms that might have resulted if the product had been so alternatively designed.

The final type of product defect is the *failure to provide* warnings of product risks or to provide adequate instructions for the product's safe use. The difference between a warning and an instruction for safe product use is that a warning merely discloses the hazards of using a product. In some circumstances, the risk of these hazards cannot be decreased or avoided, and the product seller's obligation is fulfilled once he or she has identified them and given the user the option of accepting the risk or avoiding the product. In other circumstances, however, the risks can be reduced or eliminated by safe use. In such circumstances, the seller's responsibility extends to providing instructions that will guide the user in managing the product's hazards.

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[22] Id. at 21.

[23] Restatement (Second) of Torts § 402A comment i (1965).

[24] W. Prosser, supra note 1, § 99 at 659-60.

In assessing the adequacy of the warnings and instructions provided with the product, a jury will typically be asked to consider a number of factors. The most important of these is the seriousness of the harm that may potentially result from product use or exposure. When that potential harm is great, a precise warning is generally required, even if the probability of harm is remote.

A second factor is the utility of the warning. If a significant proportion of potential users will benefit from a warning or instruction styled in a particular way, such as by using international symbols or Spanish language, the duty to utilize that style is more likely to be imposed.[25] Finally, when a manufacturer or seller has made representations concerning the safety of his or her product or aggressively promoted its use, the duty to warn of product dangers will be met only if the warnings and instructions adequately balance the effects of such representations or promotion.

The adequacy of warnings and instructions in a particular circumstance will depend, in part, on the expertise and sophistication of the product's users. In one case, for example, a worker was burned when she inadvertently brushed her face with a hand that had been contaminated by a caustic chemical resin. A Federal appeals court ruled that the adequacy of the warning must be judged from the point of view of the worker, who had limited work experience and was unaware of the specific characteristics and constituents of caustic chemicals.[26] By contrast, a different Federal appeals court in another case ruled that, because the chemical at issue was distributed only to industrial users, the manufacturer was entitled to rely on the professional knowledge and expertise of expected users in formulating warnings and instructions. The court held that the manufacturer need not warn of product dangers commonly known in the trade of which the plaintiff was a member.[27]

While the duty to warn normally arises at the time of manufacture or sale, there is a small body of case law that imposes an additional duty thereafter.[28] In these cases, courts have required sellers to make reasonable efforts to learn of product hazards and to inform product users of these risks. These decisions are likely to be especially important to persons who are exposed to chemical substances in the workplace, in light of the rapidly expanding evidence of reproductive health hazards or other toxicity associated with some of these substances. Even when a product has unavoidable hazards that are discoverable only after its sale, the product seller may have an obligation to warn about those dangers when they are discovered.

State-of-the-Art Defense

In cases where liability is alleged to be based on a product's defectiveness, the plaintiff may base his or her claim on either the negligence or product liability theories, or both. In either case, the defendant may attempt to answer the plaintiff's claim by asserting the "state-of-the-art" defense.

This defense is based on the rationale that a defendant should not be held responsible for a product-related injury when the defendant acted in compliance with the industrial state-of-the-art at the time of the plaintiff's exposure and had no legal duty to exceed the state-of-the-art. The definition of state-of-the-art is therefore critical, but the law is confused on this point, as various State courts have defined the term differently. Among the various definitions in use are:

- industry custom and practice,
- industry voluntary standards,
- government standards,
- what is practical or feasible for industry,
- the highest or most advanced form of industrial practice, and
- technical knowledge available at the time.[29]

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[25] See Hubbard-Hall Chemical Co. v. Silverman, 340 F.2d 401 (1st Cir. 1965).

[26] Billian v. Minnesota Mining & Manufacturing Co., 623 F.2d 240 (2d Cir. 1980).

[27] Martinez v. Dixie Carriers, Inc., 529 F.2d 62 (1st Cir. 1981).

[28] See, e.g., Wooderson v. Ortho Pharmaceutical Corp., 235 Kan. 387, 681 P.2d 1038 (1984) (manufacturer of oral contraceptive held to have a continuing duty to warn medical profession of dangerous side effects of which it knows or should know based on its expertise in the field, research, case reports, and scientific developments and publications).

[29] Practicing Law Institute, Occupational Disease Litigation (1983); Practicing Law Institute, Toxic Substances Litigation (1982). See also Spradely, Defensive Use of State-of-the-Art Evidence in Strict Products Liability, 67 Minn. L. Rev. 343, 344-47 (1982).

The courts of most States hold that the industry custom is "relevant but not controlling" in a tort case, because courts have generally been skeptical about using prevailing practices in industry as a measure of responsibility.[30] For example, if the prevailing practice in a particular industry is to permit unrestricted access to hazardous materials, or to fail to provide personal protective equipment to workers at risk of hazardous exposures, most courts would refuse to rule that compliance with such casual industry standards is sufficient to avoid liability, although evidence of the industry's practices could be considered by the jury.

Most States recognize a state-of-the-art defense based on the limits of technical or economic feasibility or practice, even in product liability cases, because of their reluctance to impose liability on a defendant who carefully designed, manufactured, and labeled a product only to discover a previously unknowable product defect after the plaintiffs have been injured.[31] Some States, however, do not allow the state-of-the-art defense to be asserted in product cases because the defendant's fault or negligence is not considered a relevant issue. In a landmark decision, the New Jersey Supreme Court applied this approach to toxic tort failure-to-warn suits, saying,

> Essentially, state-of-the-art is a negligence defense. It seeks to explain why defendants are not culpable for failing to provide a warning . . . But in strict [products] liability cases, culpability is irrelevant. The product was unsafe. That it was unsafe because of the state of the technology does not change the fact that it was unsafe. Strict liability focuses on the product, not on the fault of the manufacturer.[32]

The court justified its holding by rationales of cost-spreading and accident avoidance.[33] *Cost-spreading* would theoretically occur if the company was held liable, since the company could adjust the prices of its products to cover the costs of liability, thereby spreading the costs of dangerous products among all users. By contrast, if the company was not liable, the innocent victim would be unfairly forced to bear all of the economic burden of the injury from a dangerous product. *Accident avoidance* could be enhanced if imposition on industry of the costs of failure to discover hazards provides an incentive for greater safety research. It is possible, however, that the opposite result could ensue. Industry could reason that even if it were to push research and enhance the state-of-the-art, it would still be held to the standard of the state-of-the-art at the time of trial rather than the time of manufacture, so that rapid changes in the state-of-the-art would be of no benefit and consequently would provide no incentive to try to improve safety.[34]

Since this decision, the New Jersey court has retreated somewhat from the absolute liability approach. The defendant may be permitted to prove that the product's dangers were unknown and unknowable given the state-of-the-art at the time of manufacture.[35]

Fraud

A leading commentator on the law of torts has decried "the indiscriminate use of the word 'fraud,' a term so vague that it requires definition in nearly every case."[36] The accepted legal term for intentional tortious misrepresentation is "deceit" and has five principal elements:

1. a *false representation of fact*, made by the defendant;
2. *knowledge or belief* on the part of the defendant that the representation is false;

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[30] See, e.g., Texas & Pacific Ry. v. Behymer, 189 U.S. 468, 470 (1903) ("What usually is done may be evidence of what ought to be done, but what ought to be done is fixed by a standard of reasonable prudence. . . ."); Estate of Spinosa v. International Harvester Co., 621 F.2d 1154 (lst Cir. 1980) (compliance with custom does not relieve manufacturer of liability as a matter of law in a negligence case); Virginia Electric & Power Co. v. Carolina Peanut Co., 186 F.2d 816 (4th Cir. 1951) (custom pertinent on jury issue of due care); George v. Morgan Construction Co., 389 F. Supp. 253 (E.D. Pa. 1975) (custom should never be conclusive); Pan American Petroleum Corp. v. Like, 381 P.2d 70 (Wyo. 1963) (conformity to custom is not in itself the exercise of due care).

[31] Poland v. Beard-Poulan, 483 F. Supp. 1256 (W.D. La. 1980).

[32] Beshada v. Johns-Manville Products Corp., 90 N.J. 191, 447 A.2d 539 (1982).

[33] 90 N.J. at 207, 447 A.2d at 548.

[34] K. Trousdale, Industry Custom and Usage as a Defense in Toxic Tort Cases. Boston Univ. Law School (Apr. 1, 1983) (unpublished paper).

[35] O'Brien v. Muskin Corp., 94 N.J. 169, 463 A.2d 539 (1982); Feldman v. Lederle Laboratories, 97 N.J. 429, 479 A.2d 374 (1984). For a discussion of these cases, see Birnbaum and Wrubel, The N.J. Supreme Court Breathes New Life Into State-of-the-Art Defense, Nat'l. L.J., Sept. 17, 1984, at 22-23.

[36] W. Prosser, supra note 1, at 684.

3. an *intention* to induce the plaintiff to rely on the misrepresentation in taking action or refraining from taking action;
4. *justifiable reliance* on the representation on the part of the plaintiff, in taking or refraining from taking action; and
5. *damage* to the plaintiff, resulting from such reliance.[37]

Workers have sometimes successfully circumvented the exclusivity provisions of workers' compensation laws by claiming that their employers intentionally misrepresented the hazards of their workplace or concealed the true nature of these hazards. Faced with such allegations, courts have occasionally been willing to allow a tort action to proceed, under the intentional conduct exception to the exclusivity rule.[38]

Actionable deceit in a toxic exposure case may be more readily alleged than proven, however. As the list of elements indicates, an action for deceit must be based on a false representation—either express or implied from silence—concerning the hazard at issue, as well as the employer's (or other party's) knowledge that the representation is false. For example, in one Pennsylvania case, the plaintiff proved that, following an illness diagnosed as being related to her workplace use of carbon tetrachloride, she asked her employer to provide her with an alternative cleaning solvent for her use on the job. The employer falsely represented that this had been done, and the worker suffered additional illnesses as a result of her continuing exposure. The court awarded damages on the basis of these facts.[39]

In most cases, however, workers will be unable to allege that their employer misrepresented the identity of the substances to which they were exposed. Rather, the more usual allegation will be that the employer falsely represented the workplace to be safe, or that the employer intentionally concealed the nature of the worker's illness.[40] Clearly, in a case involving reproductive health hazards, where the level of technical uncertainty is often substantial, proof that the employer knew a product or exposure to be unsafe will be difficult to muster. Nevertheless, the worker may be able to prevail if the conduct, though not actually fraudulent, has all of the actual consequences and legal effects of actual fraud. This theory is known as *constructive fraud*.

Breach of Warranty

A lawsuit may be based on the defendant's breach of a contractual promise (warranty) to the plaintiff. For example, a plaintiff-employee might claim that the defendant manufacturer explicitly or impliedly represented a product to be safe for normal use and that this was part of the inducement for plaintiff to purchase and use the product. If the defendant made such a representation knowing it to be false, the plaintiff might, as has been noted, have grounds to sue for fraud. If, however, there is no evidence of either the defendant's knowledge of the danger or intention to induce the plaintiff's reliance on representations of safety (see elements 2 and 3 in the preceding discussion of fraud), the plaintiff may nevertheless claim that the defendant's actions resulted in a breach of the defendant's contractual promise to the plaintiff. Actions for breach of warranty are increasingly rare because product liability theory is almost always more favorable to plaintiffs. Product liability theory does not require a plaintiff to prove the existence of a contractual relationship or the terms of the agreement. In addition, many courts only permit breach of warranty plaintiffs to prevail if they prove the reasonable foreseeability of the injury at the time of contract, whereas such evidence is not required in product liability cases.

Prenatal Torts

The rights of the fetus in the area of tort recovery have changed dramatically over the last 40 years. Where once there was complete denial of any rights, the courts now grant recovery in almost every situation involving an injury to a viable fetus. The extent of these legal rights varies greatly among jurisdictions, however, as courts struggle with the unique problems posed by the unresolved legal status of the fetus. Although all States now recognize a right to bring an action for prenatal injuries, many jurisdictions will deny recovery unless the fetus has reached the stage of viability when it is injured.[41] In these jurisdictions, lawsuits for many injuries caused by reproductive health hazards, such as birth defects resulting from chromosomal aberrations or embryotoxicity, would not be permitted because the injury occurred prior to the viability stage.

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[37] Id. at 685-86.

[38] M. Baram, Reproductive Hazards in the Workplace: Tort Liability Law (May 1984) (unpublished report).

[39] Sumski v. Sanquoit Silk Co., 66 Lack. Jur. 118 (Pa. Comm. Pl. 1965).

[40] See, e.g., Johns-Manville Products Corp. v. Contra Costa Superior Court, 27 Cal. 3d 465, 612 P.2d 948, 165 Cal. Rptr. 858 (1980).

Until recently, courts refused to recognize a cause of action on behalf of a fetus for prenatal torts, on grounds that a fetus was not an independent biological entity to whom a duty was owed.[42] Fetal damage was regarded as an injury to the mother only, and she alone was allowed to recover for such damage. Today, all States permit at least some actions for prenatal injury, and recognize the right of a surviving infant to sue in tort for injuries sustained in utero.[43]

Viability[44]

With the discovery of the fetus' ability to survive outside the uterus at some point prior to the end of the normal 9-month gestation period, courts began to use the concept of viability to determine the point at which a fetus is owed an independent duty of care.[45] The justification for using viability as the tort liability determining point was that a fetus who could sustain life independent from the mother should not be treated like a part of its mother. Most courts, while not actually considering recovery for a nonviable fetus, have stated that only the viable fetus may recover. However, many of these courts, when actually faced with this problem, have allowed recovery for the fetus even though the injury occurred before the fetus was viable.[46]

The viability distinction has proven difficult to apply, however, in part because of medical uncertainty of the viability concept. (See *Roe* sidebar, below.) Moreover, the earliest stages of gestation may be a time of significant potential harm to a developing embryo/fetus and the period during which catastrophic prenatal injuries could occur. This suggests that the existence of liability for torts only after the fetus has become viable is based on an essentially arbitrary distinction in the case of developmental health hazards. Finally a child who is born with a birth defect is equally injured whether the injury occurred before or after viability. From both a scientific and legal standpoint, therefore, reliance on the viability distinction appears to be increasingly untenable and the trend appears to be awway from using viability as a criterion for recovery.[47]

Because the right to recover damages for fetal injury belongs to the child and not to the parent, liability to the fetus for prenatal harm is generally conditioned on the fetus' subsequent live birth. If the fetus is lost, the mother can collect for her own physical injuries, including the fetal loss. In addition, while a majority of jurisdictions allow recovery for prenatal injuries sustained at any point after conception,[48] some States still limit the cause of action to injuries sustained after viability.[49]

Although the right of a fetus to sue for prenatal injury is generally conditioned on its live birth and survival, where the fetus dies before or after birth as a result of injuries sustained in utero, a wrongful death action may also be brought by the parents in most States.[50]

The right to bring an action for wrongful death is a statutory right not recognized at common law. The view of the majority of States is that the wrongful death statutes create a new cause of action and do not provide merely for the survival of the cause of action previously possessed by the deceased. A number of States have the latter type of statute, known as a survival statute, and some jurisdictions have both wrongful death and survival statutes.[51] One court explained the difference between wrongful death and survival statutes as follows:

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[41] Note, Tort Recovery for the Unborn Child, 15 J. Fam. L. 276 (1977).

[42] Dietrich v. Inhabitants of Northampton, 138 Mass. 14 (1884).

[43] See Huskey v. Smith, 289 Ala. 52, 265 So. 2d 596 (1972) (Alabama becoming the last State to allow a cause of action for prenatal injuries); Annot., 40 A.L.R. 3d 1222, 1230 (1971 & Supp. 1983).

[44] Viability generally connotes a fetus that has reached 1,000 grams in weight and 28 gestational weeks.

[45] Bonbrest v. Kotz, 65 F. Supp. 138 (D.D.C. 1946).

[46] Note, Tort Recovery for the Unborn Child, supra note 41.

[47] See generally Note, Tort Recovery for the Unborn Child, supra note 41; Note, Pre-conception Negligence: Reconciling an Emerging Tort, 67 Geo. L.J. 1239, 1246-50 (1979).

[48] See W. Prosser, supra note 1, § 55 at 334.

[49] See Panagopolous v. Martin, 295 F. Supp. 220 (S.D. W. Va. 1969); Wendt v. Lillo, 182 F. Supp. 56 (N.D. Iowa 1960).

[50] See Note, Wrongful Death and the Stillborn Fetus: A Common Law Solution to a Statutory Dilemma, 43 U. Pitt. L. Rev. 819, 821 n.15 (1982); Note, Tort Recovery for the Unborn Child, supra note 41; 84 A.L.R. 3d 411 (1978 & Supp. 1983).

> An action under the survival statute is one for injury to the person of the deceased, and is in behalf of his estate; whereas an action under the wrongful death statute is for pecuniary loss sustained by the surviving spouse and children (or next of kin) of the deceased and is solely for their benefit.[52]

The reasoning used by the various State courts in considering whether the fetus is a person within these statutes varies because of the difference in interpretation of their wrongful death and survival statutes. In applying the statutes, the courts have been presented with four basic factual situations involving the injury and death of a fetus: 1) a viable fetus is injured, born alive, and dies; 2) a nonviable fetus is injured, born alive, and dies; 3) a viable fetus is injured and stillborn; and 4) a nonviable fetus is injured and stillborn.[53] The courts treat these situations differently:

1. If a viable fetus is injured, born alive, and dies, the courts generally allow recovery under wrongful death statutes. This is the typical application of the viability standard.
2. In at least two cases where a nonviable fetus was injured, born alive, and died, the courts allowed recovery.[54]
3. The most controversial of the wrongful death situations occurs when a viable fetus is injured and stillborn. Most jurisdictions allow a wrongful death action on behalf of a stillborn fetus if the injuries causing fetal death were sustained after viability.[55] The majority of jurisdictions considering this situation have held that a fetus is a "person," "child," or "minor child" under the jurisdictions' various statutes. A significant minority do not allow wrongful death actions on behalf of stillborn fetuses at all, regardless of the stage of development at which the prenatal injury occurred.[56] In these cases, however, the parents retain the right to sue for their own injuries, including the loss of the fetus.
4. There is only one reported decision granting recovery where a nonviable fetus was injured and stillborn: a 1955 Georgia case,[57] in which the court held that an action for death was permissible if the fetus was "quick," that is, able to move in its mother's womb. Another court, faced with the issue, declared, "If Michigan is to become the first jurisdiction to allow recovery under the wrongful death act on behalf of an unborn 3-month-old nonviable fetus, it is a determination for the Legislature."[58] A Rhode Island court, in a decision allowing recovery to a stillborn fetus that was injured while viable, stated *in dicta* that the issue of viability was irrelevant.[59]

Many reasons have been cited for denying recovery for the wrongful death of a stillborn fetus. Some courts have cited the specter of fraudulent suits because of the difficulty of proving causation in wrongful fetal death cases.[60] In addition, judicial interpretation of the term "person," as used in wrongful death statutes, has sometimes precluded fetuses from coverage.[61] The U.S. Supreme Court's 1973 abortion ruling that the word "person," as used in the Constitution, does not include a fetus seems to support this rationale.[62] (See sidebar.) However, the Court's interpretation of the Constitution is not dispositive of the issue of the term's proper meaning in State wrongful death statutes.

Perhaps the strongest argument in favor of allowing a wrongful death action on behalf of a stillborn fetus is that the failure to allow the lawsuit would reward the person or company that caused the death of a fetus by allowing him to avoid the liability that would be imposed if mere injury (rather than stillbirth) had ensued.

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[51] Note, Tort Recovery for the Unborn Child, supra note 41.

[52] Hale v. Hale, 426 P.2d 681, 683 (Okla. 1967).

[53] Note, Tort Recovery for the Unborn Child, supra note 41.

[54] Wolfe v. Isbell, 291 Ala. 327, 280 So.2d 758 (1973); Torigian v. Watertown News Co., 352 Mass. 446, 225 N.E.2d 926 (1967).

[55] See 84 A.L.R. 3d 411, 432-46 (1978 & Supp. 1984); S. Speiser, Recovery for Wrongful Death, § 4.36 at 552-56 (2d ed. 1975).

[56] See, e.g., Drabbels v. Skelly Oil Co., 155 Neb. 17, 50 N.W.2d 229 (1951); Graf v. Taggert, 4 N.J. 303, 204 A.2d 140 (1964). See also 84 A.L.R. 3rd, 411, 446-53 (1978 & Supp. 1984).

[57] Porter v. Lassiter, 91 Ga. App. 712, 87 S.E.2d 100 (1955).

[58] Toth v. Goree, 65 Mich. App. 296, 302, 237 N.W.2d 297, 302 (1975).

[59] Presley v. Newport Hospital, 117 R.I. 177, 365 A.2d 748 (1976).

[60] See Speiser, supra note 55, at 557 n.10.

[61] See Kilmer v. Hicks, 22 Ariz. App. 552, 529 P.2d 706 (1974).

[62] See Roe v. Wade, 410 U.S. 113, 157 (1973).

Pre-conception Torts

Few States have recognized the right of a fetus to sue for injuries sustained as the result of a *pre-conception* tort committed against its mother.[63] In early cases, statutes of limitations were invoked to deny a child a right to recover for injuries sustained as the result of a tortious act committed against its mother many years earlier. Today, however, the statutory time bar can be avoided in all States by invoking the limitations statute's tolling provisions for minor plaintiffs (which temporarily suspend statutes of limitations until the plaintiff is of age and presumably old enough to realize he or she has a cause of action).

A more difficult obstacle to a fetus' right to sue for pre-conception injuries is the traditional legal principle that an act of negligence committed against one person, which results in injury to another person, is not actionable by the latter.[64] While this rule has been used to deny the right to bring suit, the court decisions in which a cause of action has been allowed have stressed the countervailing legal principle that for every wrong there is a remedy.[65] It has also been suggested that a child's legal right to sue for pre-conception injury derives from an independent "right" of the child to be born free of injury.[66]

The only reported cases in which a cause of action for pre-conception injury has apparently been recognized have been brought against a physician,[67] a hospital,[68] and a pharmaceutical company.[69] It has been argued that the types of defendants on whom a duty of care toward a foreseeable fetal plaintiff should be imposed must be limited in order to avoid liability for torts against all childbearing women. Doctors, hospitals, and pharmaceutical companies are seen as logical and justifiable choices for inclusion in this class.[70] It remains to be seen whether manufacturers or employers are also to be included.

Wrongful Life

A final prenatal tort to be considered is sometimes referred to as "wrongful life." A wrongful life claim does not allege that the defendant caused injury to the plaintiff, but rather that the defendant's conduct contributed to the plaintiff's actual conception and birth, with the result that the plaintiff was born with a genetic, developmental, or other shortcoming. Wrongful life suits are generally brought against physicians and hospitals, and are typically based on unsuccessful sterilization or abortion procedures, as well as other medical practices and procedures (including the failure to perform appropriate procedures) that fail to diagnose an injured fetus and alert the parents so that the parents can decide whether to abort. Because there are drugs and possibly occupational exposures that decrease the effectiveness of oral contraceptives, it is also possible to imagine that a wrongful life claim could be considered in such a situation. The underlying premise of a wrongful life claim is that abortion or lack of conception would have been preferable to the birth of the injured plaintiff. Prior to the legalization of abortion in 1973,[71] courts refused to consider abortion as a viable option and even today resist the notion that nonexistence could ever be preferable to even a severely burdened life.

At least 16 wrongful life cases have been brought in 8 jurisdictions to date.[72] The intermediate appellate courts in two of those jurisdictions have recognized the claims.[73]

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[63] See 91 A.L.R. 3d 316 (1979 & Supp. 1983). (Three States allow such a cause of action: Missouri (Bergstresser v. Mitchell, 579 F.2d 22 (8th Cir. 1978)); Oklahoma (Jorgensen v. Meade-Johnson Laboratories, Inc., 483 F.2d 239 (10th Cir. 1973)); and Illinois (Renslow v. Mennonite Hospital, 67 Ill. 2d 348, 367 N.E.2d 348 (1977))).

[64] Nevertheless, under the legal doctrine of transferred intent, intentional torts such as deceit (fraud) which are committed against one person and result in injury to another are actionable by the injured third party.

[65] See Note, Torts Prior to Conception: A New Theory of Liability, 56 Neb. L. Rev. 706 (1977).

[66] See 40 A.L.R. 3d at 1257 (1971).

[67] Bergstresser v. Mitchell, 577 F.2d 22 (8th Cir. 1978).

[68] Renslow v. Mennonite Hospital, 67 Ill. 2d 348, 367 N.E.2d 1250 (1977).

[69] Jorgenson v. Meade-Johnson Laboratories, Inc., 483 F.2d 237 (10th Cir. 1973).

[70] M. Baram, Reproductive Hazards in the Workplace: Tort Liability Law (May 1984) (unpublished report).

[71] Roe v. Wade, 410 U.S. 113 (1973).

[72] See Rogers, Wrongful Life & Wrongful Birth: Medical Malpractice in Genetic Counseling and Prenatal Testing, 33 S.C.L. Rev. 713, 717 n.23 (1982).

[73] See Curlender v. BioScience Laboratories, Inc., 106 Cal. App. 3d 811, 165 Cal. Rptr. 477 (1980); Park v. Chessin, 60 A.D.2d 80, 400 N.Y.S.2d 110 (1977), modified sub nom. Becker v. Schwartz, 46 N.Y.2d 401, 386 N.E.2d 807, 413 N.Y.S.2d 895 (1978).

The majority's rejection of wrongful life claims has rested on several grounds. Courts argue that, by asserting that he or she should not have been conceived or born, a plaintiff fails to present a legally cognizable injury.[74] The calculation of damages by comparing impaired life with nonexistence is one the courts are either unwilling or unable to make. In addition, public policy is invoked to deny the claim for fear that anyone born into adverse circumstances would have a cause of action against the party responsible for those circumstances.[75]

Arguments in favor of granting a cause of action for wrongful life focus on the plaintiff's pain and suffering due to another's actions. According to these arguments, liability should be imposed on grounds of fairness and to deter future misconduct.[76]

An important implication of recognizing wrongful life claims is the possibility of a defective child's suit against its mother for exposing the child to harm in utero or by working at a hazardous job. While an argument can be made that a pregnant woman's liberty interests are paramount to those of the embryo/fetus during at least some stages of gestational development (and, indeed, this was the Supreme Court's holding in *Roe*), at least one court has recognized and tacitly approved the possibility of fetal suits against the mother. In response, the State legislature enacted a law barring all claims by a child against its mother alleging that the child should not have been conceived or born.[77]

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[74] See, e.g., Zepeda v. Zepeda, 41 Ill. App. 2d 240, 190 N.E.2d 849 (1963) (a minor child's claim against his father for being born illegitimate denied), cert. denied, 379 U.S. 945 (1964).

[75] Id.

[76] See Curlender, 106 Cal. App. 3d at 829, 165 Cal. Rptr. at 488.

[77] See Cal. Civ. Code § 43.6 (West 1982).

INTANGIBLE INJURIES RESULTING FROM REPRODUCTIVE HEALTH HAZARDS

Whenever a reproductive harm is suffered by a worker, it is necessarily accompanied by other, intangible losses to the worker or family members. While these intangible losses are difficult to evaluate, they are nevertheless real harms and, in certain circumstances, legally cognizable. Two such intangible harms are considered here: loss of consortium[90] and emotional distress.[91]

Loss of Consortium

Loss of consortium is the legal term applied to the loss incurred by a spouse when a marital partner suffers a personal injury. Loss of consortium encompasses any diminution or impairment of marital companionship, affection, and sexual relations.

Loss of consortium is not in itself a theory of liability, but rather an element of damage in an action based on one of the theories of liability articulated above. Because suits for loss of consortium are derivative, in the sense of being occasioned by an injury to the worker, they are generally precluded (along with tort suits by the workers themselves) by workers' compensation statutes.

Nevertheless, a suit for loss of consortium may be brought in cases where the injured worker retains the right to sue by virtue of circumstances constituting an exception to the exclusivity rule (discussed in the following section). In these cases, the workers' spouse must still allege and prove negligence,[92] a product defect,[93] or some other basis of liability.

Some courts have held that a physical injury to one's spouse is an essential element of an action for loss of consortium,[94] while other courts recognize a spouse's case for loss of consortium

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[90] See Loss of Consortium Claims: Rare But Not Impossible, O.S.H. Rep. (BNA) 37 (May 1983).

[91] See Galante, When the Mind Is Hurt, 6 Nat'l. L.J., May 28, 1984, at 1.

[92] See, e.g., Rodriguez v. Bethlehem Steel Corp., 12 Cal. 3d 382, 525 P.2d 669, 115 Cal. Rptr. 765 (1974).

[93] See General Electric Co. v. Bush, 88 Nev. 360, 498 P.2d 366 (1972).

[94] E.g., Slovensky v. Birmingham News Co., 358 So. 2d 475 (Ala. App. 1978).

Roe v. Wade and Fetal Rights

The issue of fetal rights was addressed at length in the Supreme Court's landmark abortion decision in *Roe* v. *Wade*.[78] The Court held that a woman's constitutional right of privacy "is broad enough to encompass [her] decision whether or not to terminate her pregnancy."[79] Nevertheless, the Court emphasized, a State may limit the right to abort if such limitation would serve a "compelling State interest."[80]

The Court considered what State interests would be sufficiently "compelling" to justify criminal abortion statutes and discussed three possible justifications: discouraging immoral conduct, safeguarding the health of pregnant women, and protecting fetal life.

The Court quickly rejected the first justification, both because the State had not claimed it and because the courts have never considered it seriously. The second justification, concern for the health of pregnant women, grew from the historical dangers of abortion techniques. The Court examined more recent evidence that mortality for modern abortion procedures is lower than mortality for childbirth, at least when abortions are performed early in pregnancy in licensed facilities.[81] The Court concluded that a State's interest in protecting a woman's health from the dangers of abortion does not become compelling until the end of the first trimester (13th week), at which time the woman's risk of death from abortion exceeds her risk of death from normal childbirth. After that point, the State may regulate the abortion procedure "to the extent that the regulation reasonably relates to the preservation and protection of maternal health."[82] Prior to the "compelling" point, an abortion may be performed without State interference.

The Court's reasoning implies that a change in abortion-associated or maternal mortality data would affect the time at which the State's interest in the woman's health would become "compelling." Recent data indicate that abortion does not become riskier than live birth until some point between the 16th and 20th week of gestation, or well into the second trimester.[83] Thus abortion early in the second trimester may be safer than childbirth and a State's "compelling" interest would not justify legislation until later in pregnancy.[84] Recent data indicate abortion-associated mortality is declining much faster than maternal mortality. During the 5-year period following the *Roe* decision, maternal mortality in the United States declined approximately 38 percent, from approximately 13 to 8 deaths per 100,000 live births, while mortality associated with all legal abortions declined more than 85 percent, from 3.4 to 0.5 deaths per 100,000 legal abortions.[85] Such advances in medical science are the basis for arguments that the trimester analysis of *Roe* should be abandoned.[86]

The third justification, concerning the State's interest in protecting fetal life, was also discussed. The Court held that the word "person," as used in the Constitution, does not include the unborn, and therefore the fetus itself has no constitutional right to survive.[87] The Court resolved the State's interest in the fetus with regard to the biological stages of prenatal development rather than attempting a philosophical determination of when human life begins. The Court held that a State acquires a compelling interest in the potential human life of the fetus at the moment of viability, which occurs during the early third trimester.[88] After that time, a State may prohibit all abortions that are not necessary to protect the life or health of the pregnant woman.[89] The fetus' right to survive is thus never paramount to the woman's right to life or health. Furthermore, States are not constitutionally required to prohibit third-trimester abortions because fetuses are not constitutionally protected "persons."

In sum, the Supreme Court ruling in *Roe* essentially states that:

- during the first trimester, a State may not restrict abortions;
- during the second trimester, a State may restrict abortions only to the extent reasonably necessary for the protection of maternal health; and

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[78] 410 U.S. 113 (1973).

[79] Id. at 153.

[80] Id. at 154.

[81] Id. at 163.

[82] Id. at 163. For example, the State may require that abortions after the first trimester be performed in a hospital.

[83] C. Tietze, Induced Abortion: A World Review (1981).

[84] See generally King, The Jurisdicial Status of the Fetus: A Proposal for Legal Protection of the Unborn, 77 Michigan L. Rev. 1947 (1979).

[85] Tietze, supra note 83.

[86] E.g., City of Akron v. Akron Center for Reproductive Health, 103 S. Ct. 2481, 2504 (1983) (O'Conner, J., dissenting).

[87] 410 U.S. at 158.

[88] Id. at 160.

[89] Some commentators believe that the mother and viable fetus should be protected equally. See King, supra note 84.

- during the third trimester, a State may promote its interest in potential human life by restricting or even proscribing abortions, except where it is necessary to preserve the life or health of the pregnant woman.

The Court apparently concluded that the fetus had no constitutional right to life even when viable, for an abortion is still an option after the fetus is viable unless the State chooses to proscribe abortions during the third trimester. Even if the State chooses to regulate or proscribe third-trimester abortions, it apparently cannot forbid abortions when they are necessary to preserve the life or health of the pregnant woman. Thus the State's legal right to protect (or refuse to protect) potential human life and the pregnant woman's right to preserve her life and health are both always paramount to any legal right of the fetus to be born.

The resulting situation, described by some as anomalous, is that a woman may legally and without liability abort a fetus (even a viable fetus, if the State has not passed a law forbidding such abortions or if it is necessary for the pregnant woman's life or health). Yet in every State, liability attaches to a person who merely injures a viable fetus that is later born alive (even if it only lives for a few seconds), and a few States grant the nonviable fetus this same right. (In the *Porter* case, a Georgia court granted recovery notwithstanding the fact that the fetus was never born, nor even viable when lost.)

This situation suggests that although a fetus never has a constitutional right to life, it may sometimes have a statutory or common law right (the existence and application of which varies from State to State) to be uninjured if it lives, especially if the injury occurs after the fetus becomes viable. It may also have a statutory or common law right to life which may be upheld against all but the woman who carries it.

It has been suggested that this is the rational result of a series of public policy balancing tests, in which the woman's right to privacy and reproductive freedom in early pregnancy, and to health and life in later pregnancy, are superior to the fetus' right to survive, while a fetus' right to survive and be healthy may be superior to any other person's right to interfere wrongfully with the fetus' life or health and to avoid payment of damages for the injury.

predicated on a mental or emotional injury to the other spouse.[95] In either case, a loss of consortium suit could clearly result from reproductive harm to a worker if the exclusivity rule does not apply.

Emotional Distress

Emotional distress can result from an occupationally induced physical injury (e.g., miscarriage, sexual dysfunction, sterility, or a birth defect) or even the fear of being injured by a workplace exposure. Toxic tort actions alleging psychic injury from the fear of reproductive or other harms are increasingly common.[96] The worker, the worker's spouse, the impaired child, even the worker's extended family can all suffer serious emotional effects.

The traditional legal view of emotional distress has been that such losses were not compensable unless they accompanied some physical injury and were, in turn, manifested by some physical consequence or accompanying physical illness.[97] For example, a plaintiff seeking damages for emotional distress arising out of exposure to a reproductive hazard would have to show that exposure to the hazard had resulted in some physical injury, even if only a nominal injury, in order to recover. The plaintiff would then have to present further evidence of some objective symptoms of emotional distress, such as sleeplessness.

More recently, most courts have recognized intentional infliction of emotional distress as grounds for bringing suit, even when no physical injury occurred.[98] In addition, negligent infliction of emotional distress is now recognized as an independent cause of action in eight States.[99]

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[95] Molien v. Kaiser Foundation Hospitals, 27 Cal. 3d 916, 616 P.2d 813, 167 Cal. Rptr. 831 (1980); Agis v. Howard Johnson Co., 371 Mass. 140, 355 N.E.2d 315 (1976).

[96] Galante, supra note 91, at 28. See generally Note, Increased Risk of Cancer as an Actionable Injury, 18 Ga. L. Rev. 563 (spring 1984).

[97] See generally W. Prosser, supra note 1, § 12 at 49-62.

[98] W. Prosser, supra note 1, at 52.

[99] Galante, supra note 91, at 28.

Moreover, in 1980, California became the first major jurisdiction to allow recovery for emotional distress when the plaintiff could present no physical evidence of the psychic injury.[100] Most States still require some objective symptoms, however, before they will consider emotional distress to be compensable.[101]

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[100] Molien v. Kaiser Foundation Hospitals, 27 Cal. 3d 916, 616 P.2d 813, 167 Cal. Rptr. 831 (1980).

[101] See, e.g., Payton v. Abbott Laboratories, 386 Mass. 540, 437 N.E.2d 171 (1982).

SUITS AGAINST EMPLOYERS: THE EXCLUSIVITY RULE, REVISITED

In most States, the statutory exclusivity rule of the workers' compensation statute has been construed as a bar to common law and wrongful death actions against the employer by the injured worker, the spouse of the injured worker, and the worker's dependents and children in being at the time of the worker's injury. Thus tort claims by the worker, spouse, and existing children against the employer will fail in most States due to the exclusivity rule[102] unless the plaintiff can claim and prove that the case comes within an exception to the rule. Various exceptions and limitations on the scope of the exclusivity rule have been defined by the courts and legislatures in some States, and one can discern a recent trend of uncertain strength to permit loss of consortium actions by the spouse of an injured worker, despite the rule.

Whether the exclusivity rule will be applied to bar tort suits by the fetus or impaired child or descendants, born or conceived after the worker's injury, is an open question. Because exclusivity provisions generally refer to, or have been interpreted as being applicable to, excluding tort suits by workers, spouses, and children in being and do not mention suits by future children, it can be argued that the exclusivity rule does not apply to the unborn and unconceived. Injuries to the unborn can be viewed as consequential injuries similar to the loss of consortium or emotional distress suffered by the spouse, and therefore might be barred by the exclusivity rule in most States. Yet, courts that want to refuse to extend the exclusivity rule to such cases may be able to construe narrowly the relevant statutory language or legislative intent, or depart from the view that such injuries are merely consequential to the worker's injury, because they involve breach of an independent duty by the employer to the injured fetus, child, or descendant. This view would also be supported by the fact that State compensation laws do not provide a benefit schedule for this type of loss.

At present, the exclusivity rule will usually bar tort suits against employers for reproductive injury by workers, spouses, and dependents unless some legal argument can be used to pierce the exclusivity veil. The following discussion focuses on two principal arguments that have proven effective in worker suits against employers in some jurisdictions: dual capacity and intentional tortious conduct.

Dual Capacity Exception

This exception has been adopted by a few States to permit the worker both to secure compensation benefits and to sue the employer at common law. The exception applies when the employer caused the injury while acting in a relationship to the worker that is outside of, or in addition to, the employment relationship. Dual capacity may be said to exist when the employer is also a manufacturer of the product that caused the worker's injury[103] or provides medical services in a negligent fashion.[104]

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[102] A. Larson, infra note 106, at § 66.00. See also Williams v. Schwartz, 61 Cal. App. 3d 628, 131 Cal. Rptr. 200 (1976); Williams v. State Compensation Insurance Fund, 50 Cal. App. 3d 116, 123 Cal. Rptr. 812 (1975); Cole v. Dow Chemical Co., 112 Mich. App. 198, 315 N.W.2d 565 (1982).

[103] See, e.g., Mercer v. Uniroyal, Inc., 49 Ohio App. 2d 279, 361 N.E.2d 492 (1977) (truck driver injured by tire blowout was permitted to sue employer as manufacturer of a defective tire).

[104] D'Angona v. Los Angeles County, 27 Cal. 3d 661, 613 P.2d 238, 166 Cal. Rptr. 177 (1980) (hospital worker suffering from work-related disease was permitted to recover for negligent medical treatment by the hospital-employer).

Dual capacity thus redresses the inequity of a situation where the rights of an injured worker to recover under the common law would otherwise depend on the identity of the provider of defective goods or services. Under the exception, the employer can be sued and held liable at common law for independent duties it owes to employees in its other, nonemployer, capacity.[105] Under the dual capacity exception, a company that manufactures a product posing a reproductive hazard would be equally subject to liability to its own injured employee as it would be to the injured employee of another company that uses the hazardous substance in its own production process.

This exception has been strongly opposed by industry, and has been rejected in 23 States. Nevertheless, California, Ohio, and a few other industrial States have adopted the exception to permit suits against employers under product liability theory when the employer also acts as the manufacturer, seller, or distributor of the defective workplace product.[106]

Application of the exception to employers who provide medical services has not suffered the same rejection experience, and may be increasingly important. The favorable case law to date involves only hospital or physician employers who provide medical services to employees as well as to the public, but could provide a basis for permitting suits by injured workers against industrial employers that have medical benefits programs and are now beginning to engage in screening, biological monitoring, or medical surveillance of employees.[107]

In addition, suit may be brought in some States by the worker or his or her family against individual officers or consultants of the employer firm for breach of a particular duty they owed the worker. This is not a true exception to the exclusivity rule, since it involves a third party with an independent duty the breach of which is not subject to workers' compensation law. Instead, it constitutes an option for the worker to pursue a common law action, despite the exclusivity rule, against a member of his or her employer's firm. So far, this option has been permitted primarily where the worker is injured by the negligence of a corporate physician, or independent medical personnel hired by the employer to provide medical examinations in a consulting capacity.[108]

A physician's failure to diagnose a worker's illness accurately, to treat the patient appropriately, or to carry out any other legal obligations of a physician to a patient can thus provide the basis of a tort suit against the physician.[109] State courts are divided on this issue, however, with some holding that a doctor-patient relationship exists as a matter of law (i.e., the law deems the relationship always to exist) between a corporate physician and an employee, while others disagree, and at least one court has decided that a doctor owes a duty to disclose certain medical information to an employee even in the absence of a physician-patient relationship.[110]

Physicians can seek to dismiss such suits on the ground that they are "fellow employees" who enjoy the immunity from tort suits afforded by workers' compensation law. But some courts have rejected this contention, on the rationale that the physician is more of an independent contractor than an employee. The rationale for this conclusion is that the employer is unable to fully control the physician's work, which is regulated by State medical licensure and other laws establishing the autonomy of a physician's functions and the duties owed by a physician to a patient.[111]

Finally, a few courts have found that the employer itself, when in possession of medical information as to an employee's ill health or risk of ill health, owes an independent and affirmative duty to inform the worker. Failure to carry out this duty in these cases has led to employer tort liability, overriding the exclusivity rule.[112]

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[105] See Note, Workers' Compensation: The Dual Capacity Doctrine, 6 Wm. Mitchell L.Rev. 813 (1980).

[106] A. Larson, The Law of Workmens' Compensation § 72.81-72.83 (Desk ed. 1983). In addition to Mercer, supra note 103, see Kohr v. Raybestos-Manhattan, Inc., 522 F. Supp. 1070 (E.D. Pa. 1981); Bell v. Industrial Vangas, Inc., 30 Cal. 3d 268, 637 P.2d 266, 179 Cal. Rptr. 30 (1981). Bell marks a very significant expansion of the exception for situations where the employer is not in the business of manufacturing or selling the defective product to the public, but is found to be in a dual capacity because it was in the business of "purchasing . . . processing, preparing, testing, inspecting . . . repairing, installing, endorsing . . . licensing" the use of defective products. See also Moreno v. Leslie's Pool Mart, 110 Cal. App. 3d 179, 167 Cal. Rptr. 747 (1980); and Douglas v. Gallo Winery, 69 Cal. App. 3d 103, 137 Cal. Rptr. 797 (1977).

[107] See U.S. Congress, Office of Technology Assessment, *The Role of Genetic Testing in the Prevention of Occupational Disease* (Washington, DC: U.S. Government Printing Office, April 1983). See also M.A. Rothstein, Medical Screening of Workers (1984).

[108] See Annot., 28 A.L.R. 3d 1066 (1969); F. Baron, Piercing the Compensation Veil: Third-Party Remedies for Job-Related Injuries, in Occupational Disease Litigation 78 (S. Birnbaum ed. 1983).

[109] Annot., 28 A.L.R. 3d 1066 (1969).

[110] Id.

[111] Id.

Intentional Tort Exception

A second exception to the exclusivity rule is provided in a large minority of States (by statute in most and by court decision in a few others) for intentional torts by employers. Under this exception, evidence that an employer's conduct manifested a deliberate attempt to injure a worker can be used by the worker to overcome the exclusivity rule and bring a tort action against the employer, since intentional injury is not the type of accidental workplace injury contemplated by workers' compensation law.[113]

This exception has met with slow and narrowly defined acceptance by the courts in States where it is not statutorily prescribed, and courts adopting the exception have usually set very high standards of proof, requiring the employee to show that the employer acted with "actual, specific, and deliberate intent to injure" the worker.[114] Thus, in most States, recklessly endangering an employee is not enough to create tort liability for an employer, and an employer who has knowledge of an occupational disease hazard but fails to warn the employees at risk, or who in fact fraudulently misrepresents the safety of the workplace (e.g., by removing warning or use labels from hazardous substances), is still protected by the exclusivity rule and escapes tort liability.[115]

Several recent cases indicate, however, that some courts are reducing the standards of proof and are liberalizing the definition of intentional injury to permit worker tort suits against employers. Employers have been sued for fraudulently concealing the nature and extent of the worker's occupationally caused injuries, when such concealment aggravated the worker's condition;[116] for failing to warn workers of a known disease hazard and not reporting the known hazard as required by law;[117] for deliberately removing safeguards from the workplace (or failing to install them), which had been previously installed (or required) to comply with OSHA health or safety requirements;[118] and for fraud and conspiracy to deceive workers about employment hazard conditions.[119] In addition, courts in California and a few other States have refused to bar worker tort actions against the employer for the intentional infliction of emotional distress, but in some cases have limited the exception to cases that do not involve compensable physical injuries.[120] These decisions reflect an increasingly accepted assumption that employers "in the business" of working with toxic hazards should know about such hazards, and that ignorance is the result of deliberate inattention.

This liberal trend is valuable to workers who have suffered reproductive injuries, since few cases involving reproductive injury can be expected to meet the narrow criterion that an intentional tort must involve strong evidence of a direct intent to injure, and not merely carelessness, callousness, or recklessness.

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[112] Id. See also Union Carbide Co. v. Stapleton, 237 F.2d 229 (6th Cir. 1956); Coffee v. McDonnell-Douglas Corp., 8 Cal. 3d 551, 503 P.2d 1366, 105 Cal. Rptr. 358 (1972); Annot., 69 A.L.R. 2d 1213 (1960).

[113] E.g., Barnes v. Chrysler Corp., 65 F. Supp. 806 (N.D. Ill. 1946); Le Pochat v. Pendleton, 187 Misc. 296, 63 N.Y.S.2d 313 (1946).

[114] Note, Exceptions to Exclusive Remedy Requirement of Workers' Compensation Statutes, 96 Harv. L. Rev. 1641 (1983).

[115] E.g., Phifer v. Union Carbide Corp., 492 F. Supp. 483 (E.D. Ark. 1980) (failure to warn employee of hazard or provide protective clothing held not to rise to the level of intentional conduct required to invoke exception to exclusivity rule); Kofren v. Amoco Chemicals Corp., 441 A.2d 226 (Del. 1982) (claim against employer for deceiving employee as to truth about hazards of asbestos exposure was barred by exclusivity rule).

[116] E.g., Johns-Manville Corp. v. Contra Costa Superior Court, 27 Cal. 3d 465, 612 P.2d 948, 165 Cal. Rptr. 858 (1980).

[117] E.g., Blankenship v. Cincinnati Milacron Chemicals, Inc., 69 Ohio St. 2d 608, 433 N.E.2d 572, cert. denied, 459 U.S. 857 (1982).

[118] Mandolidis v. Elkins Industries, Inc., 246 S.E.2d 907 (W.Va. 1978) ("deliberate intention" was not limited to intent to kill or injure, but included conduct constituting an intentional tort or willful misconduct).

[119] McDaniel v. Johns Manville Corp., 487 F. Supp. 714 (N.D. Ill. 1978).

[120] McGee v. McNally, 119 Cal. App. 3d 891, 174 Cal. Rptr. 253 (1981).

DEFENDANTS

Potential Defendants

It is common practice for a plaintiff's attorney to name all plausible defendants in a tort action, thereby forcing each defendant to come forward with a legal or factual basis for exculpation. By naming all of these defendants in a single lawsuit rather than filing one lawsuit for each defendant, the plaintiff can optimize his or her chances for recovering against one or more defendants, and avoid the possibility that the juries in separate proceedings will reach inconsistent results.

Negligence

In a reproductive hazard lawsuit in which negligence is alleged, the list of potential defendants obviously begins with those responsible for the existence of the hazard. While one's employer and fellow workers may enjoy immunity for their negligence under the applicable workers' compensation law, others who are responsible for a hazard may not enjoy similar immunity. These parties may include workplace design engineers or architects,[121] outside safety or insurance consultants or inspectors,[122] or the owner of the premises (other than one's employer) at which work is taking place.[123] Any of these persons may have been negligent in creating or evaluating the workplace hazard, and thus may be liable for negligence if they failed to exercise ordinary care in the provision of their professional services.

Similarly, others (including company physicians) who could have prevented or ameliorated a reproductive harm may be held liable for negligently failing to do so, as noted in the section on dual capacity.

Strict Liability and Product Liability

The least burdensome evidentiary requirements exist for strict and product liability suits because the defendant's negligence need not be proven. Hence, persons arguably engaged in abnormally dangerous activities and commercial sellers of products are potentially important defendants in a tort action. The former category might include, for example, the operator of a hazardous waste facility, who may be strictly liable for reproductive harms to workers other than its own employees who come on to the premises to deliver waste or to transact other business. (Employees of the facility would be subject to the exclusivity rule.) The category of sellers would include all commercial sellers, beginning with the manufacturer, and including wholesalers, distributors, and retailers. In some circumstances, repairers, installers, construction contractors, and rebuilders might be deemed to be sellers if they deliver products to buyers in the course of rendering services.[124]

Deceit

A party who engages in intentional deceit may also be named as a defendant in an action arising out of reproductive harm, regardless of whether the person actually created the hazard in question.[125]

Multiple Defendants

In some cases, tortious conduct by separate defendants might have led to a reproductive injury that would not have occurred but for the concurrence of separate acts. In an exaggerated example: a manufacturer produces a dangerously contaminated chemical product, an independent quality control inspector unreasonably fails to discover the contamination, a distributor sells the chemical to the employer of a particular worker, a second manufacturer makes defective personal protective equipment and sells it to the employer, the employer knows that the personal protective equipment is defective but represents to the employee that it is functional, and a physician negligently fails to diagnose the employee's uptake of the dangerous chemical.

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[121] Comment, Recent Statutory Developments Concerning the Limitations of Actions Against Architects, Engineers and Builders, 60 Ky. L.J. 462, 465 (1972).

[122] Kohr v. Johns Manville Corp., 534 F. Supp. 256 (E.D. Pa. 1982).

[123] E.g., Mahoney v. J.C. Penney Co., 71 N.M. 244, 377 P.2d 663 (1963).

[124] Toledo P. & W.R.R. v. Burlington N., inc., 67 Ill. App. 3d 928, 385 N.E.2d 937 (1978).

[125] Garcia v. Halsett, 3 Cal. App. 3d 319, 82 Cal. Rptr. 420 (1970).

Whenever more than one defendant is named in a tort action, the question arises as to how to best apportion responsibility among the various defendants. It is often impossible to establish the precise contributions of multiple independent factors to their injuries. Because of this problem, many jurisdictions have adopted the *substantial factor rule,*[126] which states that any defendant whose activity was a substantial factor in bringing about the plaintiff's injury can be held liable for the entire injury. In the hypothetical example cited, all six defendants may be held liable for the employee's injuries. Thereafter, if the court finds a reasonable basis for apportioning liability among the defendants, it may do so.[127]

The usual mechanism for apportioning liability among defendants is the cross-claim, in which a defendant files a claim against another defendant, seeking either *indemnity* or *contribution.* Indemnity is the recovery from another party of the full amount of one's liability. Contribution is the recovery from another party of a portion of one's liability. As a general rule, a passively negligent defendant may obtain indemnity from one who is actively negligent.[128]

Critics of this rule have often noted that logic does not support imposition of the entire liability on a single party. It has been suggested that requiring all responsible parties to share in the loss would be more equitable. For this reason, contribution has come to dominate the allocation of responsibility among people who commit torts.[129] Although an integral aspect of contribution is apportionment by fault, it is not always clear which party is mostly at fault, as can be seen from the example discussed here.

Liability When Defendant Is Unidentifiable

A troubling problem arises when only one defendant's act was a substantial factor in bringing about the plaintiff's harm but it is impossible to identify that defendant. This situation may be especially likely to arise in reproductive harm cases involving toxic exposures, both because it may be impossible to identify which of several reproductive health hazards gave rise to the injury and because the precise commercial seller of a generic product may not be known.

The traditional legal rule applicable to such situations, known as the *alternative liability theory,* was first articulated in a 1948 case in which the plaintiff was injured by the pellet from the gun of one of two hunters who negligently fired in the plaintiff's direction.[130] Because it was clear that both hunters had exposed the plaintiff to an unreasonable risk of harm, the court shifted the burden of proof to the hunters to demonstrate who actually caused the injury. Unless one hunter proved that the other was responsible, both would be held liable and the plaintiff could recover his full damages from either.

Application of the alternative liability theory in reproductive harm cases is more complex than application of the theory to the hunting case, however. In that case, it was known that both defendants acted negligently and that one of the defendants was certainly responsible for the plaintiff's injury. In contrast, it may be impossible to place responsibility for the existence of a particular chemical in the workplace on a particular manufacturer when dozens or even hundreds of chemical manufacturers may be involved to varying degrees.

In an analogous situation, a group of DES-exposed daughters brought suit for their injuries against a number of companies that had manufactured the drug. It was unclear which of the manufacturers was responsible for each plaintiff's injuries. The court responded to the problem of allocating responsibility by creating a new legal theory, known as the *market share theory,*[131] apportioning responsibility to each manufacturer based on its share of the DES market at the time the injuries occurred. This avoided the inequitable consequences of the alternative liability theory, which could have resulted in imposing all of the responsibility on a manufacturer with only a small share of the market.

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[126] Anderson v. Minn. St.P. & S.S.M.R.R., 146 Minn. 430, 179 N.W. 45 (1920); Restatement (Second) of Torts § 431A (1965).

[127] See, e.g., Velsicol Chem. Corp. v. Rowe, 543 S.W.2d 337 (Tenn. 1976).

[128] E.g., Sargent v. Interstate Bakeries, 86 Ill. App. 2d 187, 229 N.E.2d 769 (1967).

[129] Ford Motor Co. v. Robert J. Poeschel, Inc., 21 Cal. App. 3d 694, 98 Cal. Rptr. 702 (1971).

[130] Summers v. Tice, 33 Cal. 2d 80, 199 P.2d 1 (1948).

[131] Sindell v. Abbott Laboratories, 26 Cal. 3d 588, 607 P.2d 924, 163 Cal. Rptr. 132, cert. denied, 449 U.S. 912 (1980).

The difficulty that the market share theory poses in reproductive health hazards cases is that injuries may not have been caused by exposure to a single product. Rather, the harm may be due to the additive or synergistic effects of exposures to a variety of hazards. When this is the case, the market share theory suggests that it may be most appropriate to impose partial responsibility on *each* manufacturer of *each* of the chemicals that contributed to the injury. The problem is that, although liability can easily be divided among manufacturers for a particular chemical under market share theory by examining the manufacturers' respective market shares, liability cannot easily be divided among the manufacturers of different substances.

For example, if the plaintiff is exposed to two reproductive health hazards, A and B, which have additive or synergistic effects, liability should theoretically be divided between all manufacturers of A and all manufacturers of B, based on each hazard's respective contribution to the plaintiff's injury. The liability of all manufacturers of A and B, respectively, would then be divided among those manufacturers based on each company's market share of A or B. While it may be relatively easy to identify market share, for the purpose of allocating responsibility *among* producers of A or *among* producers of B, it is not easy to identify the respective contributions of A and B to the plaintiff's injury for the purpose of dividing liability *between* makers of A and makers of B.

The Problem of Bankruptcies and Successor Corporations

In the last analysis, awards of compensation for reproductive harms are illusory if the defendant against whom the judgment is rendered is no longer in business, or if a chapter 11 bankruptcy reorganization[132] has absolved the defendant of responsibility to pay any judgment. Each of these possibilities is especially problematic in cases where injuries occur long after the time of exposure or where many similar actions are brought against a single product manufacturer.

The reorganization petition filed in Federal bankruptcy court by the Manville Corp. in 1982[133] raised for the first time the possibility that a large number of occupationally diseased workers (both Manville employees and construction industry workers exposed to Manville products) may ultimately be unable to recover the full measure of their damages from the company. Indeed, the precise purpose of the reorganization petition is to shield the corporation from the approximately 16,500 pending and 30,000 expected future lawsuits arising out of exposure to the company's asbestos products. The Manville case points out an important fact: the resources of any business enterprise are not limitless. In a case where a single manufacturer is liable for a large number of occupational or product liability injuries, corporate resources can be depleted and some of the persons injured can go uncompensated, even when they have won their cases in court.

To avoid such crushing liabilities, stockholders have sometimes dissolved an existing corporation with such liabilities and formed a new corporation to carry on the enterprise. When a new enterprise acquires an existing corporation, the assets and liabilities of the corporation are passed on to the new enterprise.[134] For this reason, a new enterprise may seek to purchase only the assets of an existing corporation, but not its stock.[135] Today, however, courts are more willing to look at the motivations of such transactions and are less inclined to allow legal responsibility to be circumvented, especially if the new enterprise is engaged in the same line of business as the old one, using the same premises and equipment, and employing many of the same people.[136]

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[132] Chapter 11 of the Federal bankruptcy statute protects a business from its creditors so that the business can continue to operate and, after it has financially recovered, pay its debts. 11 U.S.C. §§ 1101-1174 (1982).

[133] See Note, The Manville Bankruptcy: Treating Mass Tort Claims in Chapter 11 Proceedings, 96 Harv. L. Rev. 1121 (1983).

[134] Menacho v. Adamson United Co., 420 F. Supp. 128 (D.N.J. 1976); Applestein v. United Board & Carton Corp., 60 N.J. Super. 333, 159 A.2d 146, aff'd, 33 N.J. 72, 161 A.2d 474 (1960).

[135] Forest Laboratories, Inc. v. Pillsbury Co., 452 F.2d 621 (7th Cir. 1971).

[136] Shannon v. Samuel Langston Co., 379 F. Supp. 797 (W.D. Mich. 1974).

It is precisely because the courts scrutinize methods of avoiding liability that the bankruptcy strategy has proven so attractive to Manville and others who face potentially ruinous liability. Thus, chapter 11 reorganization is now being touted by some as a viable risk managment technique for risk-laden businesses.[137]

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[137] Power, Voluntary Chapter 11: A Viable Risk Management Tool?, Risk Mgmt. 19 (Dec. 1983).

LEGAL CAUSATION

Proving Legal Causation

The greatest obstacle to recovery for any reproductive harm against any of the potential defendants under any of the theories of liability is proof that exposure to one or more hazards was more likely than not a substantial factor (though not necessarily the only factor) in causing the particular reproductive injury for which monetary damages are sought. This is known in the law as *causation*, and the burden of proving it rests on the plaintiff. The requisite standard of proof is the preponderance of the evidence standard articulated in the preceding chapter.

To prove legal causation of a reproductive harm from a chemical, physical, or biological substance, the plaintiff must show the existence of a chain of events or facts which, taken together, are deemed legally sufficient to show that it is more likely than not that the plaintiff was reproductively injured by a workplace hazard. The specific events and facts to be proven will generally necessitate evidence of:

- hazardness of the substance (e.g., mutagenicity, teratogenicity, toxicity);
- emission of the substance in the workplace (e.g., levels, duration);
- plaintiff's exposure to the substance (e.g., level, duration, type of exposure);
- plaintiff's uptake of the substance (e.g., as measured in blood, urine, etc.);
- biological response after plaintiff's exposure (e.g., blood level, chromosomal change); and
- plaintiff's reproductive injury.

A plaintiff who fails to establish any one of these facts will generally lose in the tort action.

Because each of these facts may involve considerable medical and scientific uncertainty, the practical problem of proving legal causation by a preponderance of the evidence can be a formidable and costly procedure requiring the testimony of several scientific and medical experts. Since each party will have its own experts testifying in support of its contentions, a personal injury trial may become a "battle of experts," with each party attempting to convince the jury that its experts are more qualified. The need for expert witnesses in personal injury litigation has spawned an industry of experts willing to provide litigation support.

The principal expert used in tort litigation where personal injuries are at issue is the medical doctor.[138] The physician can provide expert and direct evidence pertaining to the nature of the injury (diagnosis) and its status over time (prognosis). If the personal medical history of the plaintiff is available to the doctor, or better yet, if the doctor has been the plaintiff's personal physician, the doctor may be able to provide direct evidence of the plaintiff's prior health.

This doctor, or another medical expert, may then be willing and capable of providing an expert opinion as to the missing link—causation—in the standard format required by most courts in personal injury actions. Generally, the physician does this by testifying that, based on professional qualifications, knowledge, and experience, the expert's opinion and experience leads him or her to believe that it is a "reasonable medical certainty" that the plaintiff's exposure to one or more workplace agents caused the plaintiff's injury.[139]

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[138] Mitchell, Cancer Causation and Risk in Chemical Litigation and Regulation, 3 Envtl. Analyst 6 (Nov. 1983).

[139] Id. See also Henderson, Medical Causation in Products Liability Disease Litigation, Trial 53 (June 1981); Tillevitz, Judical Attitudes Towards Legal and Scientific Proof of Cancer Causation, 3 Colum. J. Envtl. L. 344 (1977) (listing of cases in which variations on "reasonable medical certainty" were accepted).

Without this reasonable certainty, the opinion testimony will generally be excluded from the jury's consideration.[140]

The problem with using medical practitioners as the principal experts in personal injury litigation is the limited experience and perspective of most doctors. Clinical physicians are generally concerned with diagnosis and treatment, whereas biomedical researchers and epidemiologists focus more on the etiology of disease. "The definition of causation holds far more fascination for society and lawyers than it does for doctors."[141] Thus, although animal studies may show a substance to be toxic to an animal fetus, a clinician may be reluctant to draw conclusions based on animal studies alone because of the considerable species variation in effects. Doctors are also likely to stress the role of various environmental and genetic factors outside of the workplace, notwithstanding the fact that such interactions are likely to be legally irrelevant so long as the workplace exposure played a substantial role in the reproductive harm. Furthermore, few physicians are knowledgeable about occupational disease.

The testifying physician will often need scientific data in order to provide an opinion on causation; this usually requires prior testimony by one or more expert witnesses from the health sciences. It has been noted that "each toxic tort action should be regarded as a mini-research project with scientists and lawyers as co-principal investigators."[142] The testimony of a physician is deemed essential to establish or rebut causation in a particular case and is considerably strengthened by—indeed, in most cases, requires—epidemiological data, animal studies, and other scientific evidence in order to draw convincing inferences regarding the cause of a particular plaintiff's injury. But the judicial response to epidemiological and toxicological evidence has usually been skeptical. **Most courts are of the opinion that scientific evidence, by itself, is insufficient to either prove or disprove causation of a particular disease in a particular person, with toxicological evidence deemed of more limited evidentiary value than epidemiological evidence. This is based on the judiciary's concerns regarding the applicability and relevance of epidemiological and toxicological evidence to a specific individual case, and the ability to extrapolate study group results to another group which includes the plaintiff.[143] Scientific evidence is therefore viewed by the parties mainly as a set of building blocks on which a physician may rely to support an opinion on medical causation.**

To enhance the supporting roles of toxicological and epidemiological data, both plaintiffs and defendants have sought to package such scientific findings by using risk assessment modeling. The risk assessment will attempt to evaluate and quantify all factors deemed scientifically relevant, thereby generating probabilistic outcomes as to human health risk.[144] Risk assessment may be a persuasive method of packaging information relating to causation in a specific case, if the assessment or model includes the results of scientifically valid studies and considers all relevant causal elements and their interrelationships. However, this approach often runs into problems based on the model's assumptions regarding extrapolation from a study group to the plaintiff (an epidemiological issue), extrapolation from animals to humans (a toxicological issue), and extrapolation from high to low doses (a toxicological issue).[145] Other problems arise from the nature of quantitative risk assessment itself, including the issues of the quantity of data needed to create a meaningful model and whether a single model can be developed to represent all cases. Predictions made from individual models are only as good as the assumptions they contain. As one commentator noted:

> A one-hit model assumes that the risk of a particular injury from a particular substance is

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[140] Mitchell, supra note 138, at 6 (citing W. Prosser, The Law of Torts 218 (2d ed. 1955)).

[141] Mitchell, supra note 138, at 6 (citing A. Golden, Pathology: Understanding Human Disease, 108 (1983)).

[142] Epstein, The Role of the Scientist in Toxic Tort Case Preparation, Trial 38 (July 1981).

[143] See generally Hall & Silbergeld, Reappraising Epidemiology: A Response to Mr. Dore, 7 Harv. Envtl. L. Rev. 441, 442 (1983); McElveen & Eddy, Cancer and Toxic Substances: The Problem of Causation and the Use of Epidemiology, 33 Clev. St. L. Rev. 29 (1984).

[144] Shelton, Defending Cancer Litigation: The Causation Defense, For the Def. 12 (Jan. 8, 1982).

[145] See Leape, Quantitative Risk Assessment in Regulation of Environmental Carcinogens, 4 Harv. Envtl. L. Rev. 86 (1980).

> directly proportional to exposure. Halving the exposure is assumed to halve the risk. Under this assumption, no substance ever reaches a "no effect" level, but, rather, it is assumed that somewhere in a given population, some person will be so sensitive that exposure to even a single molecule of a substance could trigger an adverse reaction . . . [This] may not bear any resemblance to known scientific data, nor . . . [be] valid in pinpointing the cause . . . or even necessarily the probability of the cause . . . from low-level exposure. In fact, for most data sets, the "one-hit" model, as applied by the Cancer Assessment Group (of EPA), . . . is really designed to assure safety, and its use results in a safety factor.[146]

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[146] Id.

OTHER CONSIDERATIONS

Statutes of Limitations and Repose

State statutes of limitations and repose may limit a plaintiff's right to sue for reproductive and other injuries, due to the passage of time. *Statutes of limitations* require that a lawsuit be initiated within a specified period of time, generally 1 to 3 years, after the right to sue has accrued. In the past, the right to sue (and thus the running of the statute) was considered to begin at the time the plaintiff's injury was caused, even if the plaintiff had not yet become aware of any injury. Thus, if a surgeon negligently left a sponge in the plaintiff's chest cavity, the statute of limitations would begin to run immediately, notwithstanding the plaintiff's ignorance of the situation and lack of symptoms until several years later. The traditional application of such statutes could thus bar a plaintiff from suing.

To ameliorate the harsh effect of such a rigid time bar, most States have by statutory amendment or judicial decision adopted the *discovery rule,* holding that the right to sue and the running of the statute begin at the time the plaintiff's injury was discovered or reasonably should have been discovered.[147] For example, if a plaintiff was made sterile by an occupational exposure to a hazardous substance, and did not attempt to conceive children until some years later, most courts would begin the statutory countdown at the time the plaintiff discovered or reasonably should have discovered the injury, whichever is earlier. In the case of toxic torts, a few courts would not begin counting until the plaintiff not only discovered the fact of the injury but also discovered (or reasonably should have discovered) the causal connection between the injury and the defendant's conduct.[148]

In recent years, the widespread adoption of discovery rules has been met with counter reform measures, often proposed by manufacturers who fear unlimited liability for the life of their products or the duration of long latency periods. As a result, some States have enacted *statutes of repose* for products liability suits that require that a lawsuit be initiated within a specified period of time (generally 10 years) after the occurrence of the incident that gave rise to the injury. Statutes of repose may therefore prevent plaintiffs from suing for reproductive diseases with latency periods longer than the statutory period, as well as bar plaintiffs who fail to discover reproductive problems due to prolonged sexual abstinence or use of contraception.

The current status of State statutes of limitation and repose is one of very little uniformity, as a number of State courts have declared their State's statute of repose to be unconstitutional.[149]

Prior Litigation

Two legal principles, designed to promote the efficient use of judicial resources, may have an important impact on reproductive hazard litigation. The first of these is *res judicata*, a doctrine which holds that a judgment on the merits of a prior suit involving the same parties (or those who have certain relationships with such parties) bars another suit on the same grounds. For example, if a worker sues a product manufacturer for intentionally concealing the hazardous nature of a product, whoever loses the lawsuit may not relitigate at a future time, notwithstanding the discovery of new evidence after the trial.

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[147] See, e.g., Williams v. Borden, Inc., 637 F.2d 731 (10th Cir. 1980); Louisville Trust Co. v. Johns-Manville Products Corp., 580 S.W.2d 497 (Ky. 1979).

[148] See, e.g., Frederick v. Calbio Pharmaceuticals, 89 Cal. App. 3d 49, 152 Cal. Rptr. 292 (1979); Raymond v. Eli Lilly & Co., 117 N.H. 164, 371 A.2d 170 (1977).

[149] See, e.g., Lankford v. Sullivan, Long & Hagerty, 416 So. 2d 996 (Ala. 1982); Battilla v. Allis Chalmers Mfg. Co., 392 So. 2d 874 (Fla. 1980); Bolick v. American Barmag Corp., 54 N.C. App. 589, 284 S.E.2d 188 (1981). See also McGovern, The Variety, Policy and Constitutionality of Product Liability Statutes of Repose, 30 Am. U.L. Rev. 579 (1981).

The second doctrine is *collateral estoppel*, which applies when the second suit is based on a similar injury to a different person. Under the doctrine of collateral estoppel, the judgment in the prior suit precludes relitigation of the particular issues actually litigated and necessary to the outcome of the first lawsuit. Since certain types of suits against a single defendant or group of defendants will necessarily involve many of the same issues in each case (e.g., was the product dangerously defective?), collateral estoppel is potentially an economical device to avoid relitigating the same issues. Under the collateral estoppel doctrine, once a product has been adjudged defective or an activity ruled to be abnormally dangerous, the defendant is precluded from relitigating that issue in a later lawsuit brought by another plaintiff. The second plaintiff can dispense with evidence on the issue and proceed to the other elements of his or her case (generally, the nature and extent of the particular plaintiff's injury).

Until recently, however, the doctrine of collateral estoppel was not available for use by plaintiffs in such ways. The doctrine was limited to circumstances where "mutuality" of estoppel existed.[150] This restriction meant that collateral estoppel was unavailable unless the party seeking to invoke the estoppel would himself have been barred from relitigating the point if the prior judgment had been the reverse. For example, a product liability plaintiff would not be able to use collateral estoppel to demonstrate defectiveness since he or she would not be bound by a judgment against some other plaintiff based on a lack of a product defect.

In many jurisdictions, the mutuality of estoppel requirement has been abandoned.[151] The distinct trend of judicial authority is to permit the type of "offensive" collateral described here.[152] Only in cases where it would be unfair to the defendants (e.g., where the plaintiff could have joined a prior suit, but failed to do so to avoid the burden of an adverse judgment while using collateral estoppel to reap the benefits of a favorable judgment) will a court that does not require mutuality refuse to impose offensive collateral estoppel.[153]

Sovereign Immunity

Under the common law, the concept of the government's immunity from liability was firmly grounded in the notion that "the King can do no wrong" and could not be sued without the government's permission. Because of the involvement of the Federal Government in the inspection and certification of workplaces and the provision of information concerning reproductive health hazards, the question arises as to whether the Government can be held liable for its negligence in performing any of these functions. Under the Federal Tort Claims Act (FTCA), the United States is liable for:

> . . . any negligent or wrongful act or omission of any employee of the Government while acting within the scope of his office or employment, under circumstances where the United States, if a private person, would be liable to the claimant in accordance with the law of the place where the act or omission occurred.[154]

An exception to this rule exists: the Government cannot be held liable for "any claim arising out of . . . misrepresentation [or] deceit. . . ."[155]

The dividing line between negligence (which can serve as the basis for a lawsuit) and misrepresentation (which cannot) is not entirely clear in cases involving inspections, certifications, and failure to warn. It appears that if an inspection is conducted exclusively for the purpose of making a statement about the inspected premises (e.g., that they comply with regulations or are free from hazards), and the primary responsibility for safety continues to lie with the premises' owner or operator, then the Government's failure is alleged misrepresentation and not actionable. But if the inspection is conducted as part of a program assuring safety compliance, and the Government takes on some responsibility for assuring safety, then the claimed wrong may be negligence in inspection and is not barred.[156] In either case, Government workplace inspections are difficult bases for imposing liability on the United States because of the limited waiver of sovereign immunity by the FTCA.

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[150] See, e.g., Bigelow v. Old Dominion Copper Co., 225 U.S. 111, 127 (1912).

[151] See, e.g., Blonder-Tongue Laboratories, Inc. v. University of Illinois Foundation, 402 U.S. 313 (1971).

[152] Restatement (Second) of Judgments § 88, Reporter's Note (1982).

[153] See Parkland Hosiery Co. v. Shore, 439 U.S. 322, 331 (1982).

[154] 28 U.S.C. § 1346(b) (1982).

[155] Id. at § 2680(h).

Conclusion

Whether or not workers' compensation is made to apply to some or all reproductive harms, the tort liability system will continue to be available for recovery of damages against parties outside the system. Indeed, for workers injured by exposure to hazardous products in the workplace, both the workers' compensation system and product liability action against commercial sellers of such products will likely continue to be available as avenues of redress though opportunities to collect through either have limitations.

In light of this fact, the possibility of double recovery is raised. In some jurisdictions, this possibility is eliminated by a rule which subtracts any compensation from "collateral sources" (e.g., workers' compensation) from tort judgments. In other jurisdictions, workers' compensation benefits must be repaid if damages from the same injury are recovered from a third party such as a product manufacturer. In still others, the possibility of a true windfall recovery is regarded as so remote—because of the low levels of workers' compensation and the substantial legal fees that are paid by prevailing plaintiffs in tort actions—that no such set-off is deemed necessary. Resolution of this debate involves consideration of circumstances beyond those presented in reproductive hazard cases, however, and cannot be achieved within this more narrow context.

Considerable interest in "victim's compensation" legislation, designed to provide a speedier and more effective remedy for toxic torts, has been evidenced at both the State and Federal levels in recent years. Such legislation is designed to remedy the problems posed by the substantial barriers to recovery by toxic tort plaintiffs—particularly in the area of causation—which have been discussed here. Whether such barriers are as substantial in practice as they are in theory has not been demonstrated, however. Indeed, it is instructive to note that victim's compensation legislation is sometimes supported by industry, if it includes limitations on liability and an exclusivity rule barring tort actions against those who pay compensation under such statutes. In contrast, such legislation is typically opposed by the plaintiff's bar and consumer and environmental groups if such exclusivity provisions are incorporated and damages for intangible harms (e.g., pain and suffering) are limited. Again, however, resolution of this policy debate exceeds the scope of the reproductive harm compensation issue.

One important tort law issue that is limited in scope to reproductive harms involves the rights of the unborn to recover damages for prenatal or even pre-conceptual torts. A small trend in favor of allowing such recovery, regardless of the fetus' subsequent live birth or viability at the time of the tortious exposure, may be on a collision course with the abortion rights established in *Roe v. Wade*. The increasing recognition of the fetus' right to recovery for tortious injury may be consistent with the Supreme Court's holding that a fetus is not a "person" within the meaning of the Constitution if fetal rights are seen as subordinate to the pregnant woman's rights but superior to the rights of third-party tortfeasors.

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[156] M. Baram, supra note 38.

Chapter 11

The Ethical Issues

CONTENTS

Page
Introduction 329

The Parties at Risk 330
  Female Workers 330
  Male Workers 330
  The Embryo/Fetus 330

Moral Principles at Stake 331
  Respect for Persons 331
  Beneficence 332
  Justice 335

Chapter 11

The Ethical Issues

INTRODUCTION

Ethics requires people to think about the justifications for their actions. Ethical principles such as beneficence or justice provide the justification for action and are called into play when persons are faced with ethical dilemmas. In an ethical dilemma, mutally exclusive courses of action can each be supported by weighty moral arguments. A situation becomes a dilemma when the principles that ground the reasons or arguments in support of each course of action are important and serious, and none is in any obvious way the right set of reasons. Regardless of the course selected, one's choice will be desirable in some respects but undesirable in others. The dilemma thus presents a problem of choice between values, or among a hierarchy of values.

This chapter reviews the basic ethical principles and arguments entailed by alternative policy options with respect to reproductive health hazards in the workplace. The chapter begins by reviewing the moral position of the various parties at risk. The most important ethical principles surrounding reproductive health hazards in the workplace are then discussed: respect for persons, beneficence, and justice. The ethical dimensions of specific dilemmas in reproductive health hazards in the workplace are discussed in "Selected Ethical Issues in the Management of Reproductive Health Hazards in the Workplace," available from OTA and NTIS (see app. F). These dilemmas include acceptable risk in the workplace, discrimination and job termination, compensation for harm or damage, the right to know, and funding for research into and surveillance of reproductive health hazards in the workplace.

The prevention of reproductive harm shares many aspects of efforts to prevent occupationally linked diseases in general. But certain special characteristics of the reproductive health problem must also be acknowledged and addressed.[1]

In contrast to occupational injuries, but in common with other occupational diseases, reproductive impairment often lacks a clear link with occupational exposure. Data linking workplace exposure and a reproductive endpoint are almost always probabilistic in nature. The rate of occurrence of a particular reproductive endpoint is often low in the overall population, making it difficult to demonstrate a significant increase in a small worker population. A key feature is *uncertainty* at several levels: uncertainty about the existence of any relationship between workplace exposure and a reproductive disorder; uncertainty about the cause of any specific case of reproductive disorder; uncertainty about the strength of a relationship and its consequent level of risk. Although this inherent uncertainty cannot be denied, fair and reasonable public policies can nevertheless be formulated to deal with exposure to workplace hazards to reproductive health.

Exposure to reproductive health hazards raises new morally relevant problems in three areas. First, disputes over the existence and management of reproductive health hazards often focus on women workers, who have been made vulnerable to some of the negative consequences of special protective interests. Second, there may be the presence of an embryo/fetus, who cannot be asked for his or her consent to the risks that may be taken. Third, reproduction is one of the most sensitive and intimate aspects of human life. Securing information about reproductive health thus raises questions about the protection of individual privacy.

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[1] The introduction that follows is drawn from a contractor report prepared for OTA by Ron Bayer of the Hastings Center.

THE PARTIES AT RISK

Female Workers

Much of the scientific literature has focused solely on the risks to reproduction posed by the exposure of women to reproductive toxicants or their unborn children to developmental toxicants. The traditional approach has been a culturally informed definition of the research agenda, which is that women are the primary agents of procreation, and their exposure to potential hazards should be studied.

Efforts have consequently been made to protect female workers from exposure to toxic substances believed to pose some risk to their reproductive health. Such efforts have typically taken the form of exclusionary practices justified by concern for the health of potential offspring rather than for the reproductive health of the woman per se. Yet risk to a fetus may also be a risk to the woman herself. It may be direct, as in the risk to her own reproductive health; less direct, as in the risk to her health posed by a spontaneous abortion; or indirect, in that she may suffer psychological damage and diminished life prospects with the occurrence of a miscarriage or on the birth of a dead or damaged baby. Finally, women often risk economic loss and discrimination in the face of policies that restrict job opportunities on the grounds of protecting them from exposure to health hazards.

Male Workers

Research results are increasingly showing the vulnerability of male workers to reproductive harm. For example, mutagens are now known to affect sperm cells in ways that can compromise the viability of the embryo and fetus. Exposure to toxic substances can also cause loss of libido, disruption of hormone balance, impotence, and other adverse consequences relative to sexual and procreative function. And male workers may "bring home" toxic substances on their clothing or bodies, affecting spouses and children who would not otherwise have been exposed to reproductive harm. Male workers also share the harm caused by the birth of a dead or damaged child. However, since most protective policies are directed toward the protection of women or their pregnancies, men who go on working in a hazardous environment are more likely to be exposed to risk of harm from toxic substances.

The Embryo/Fetus

It is well known that exposure of workers to reproductive and developmental toxicants can kill an embryo/fetus or produce live offspring who are permanently harmed.

Exposure to developmental toxicants in the workplace poses a grave problem for those who view the embryo as possessing moral rights to survival from the moment of conception. Even those who disagree about the validity of such embryonic and fetal rights, however, recognize the possibility of spontaneous abortion or fetal harm as profound concerns.

Unique ethical problems surround the fetus. First, because it is voiceless and unconsenting, the fetus can have no say in decisions affecting its well-being, and cannot be assumed to consent to whatever risks it may encounter.

Second, the welfare of the fetus who eventually becomes a live-born child is ineluctably tied to the welfare of its parents. A fetus whose parents are denied work because of a perceived reproductive hazard is not necessarily better off as a result. One or both parents may be forced to accept even riskier employment elsewhere, or to remain unemployed and face the financial and psychological consequences associated with unemployment.

Third, the fetus has an ambiguous and changing moral status. The multitude of views expressed in the abortion debate underscores the apparent paradox: a grievous but nonfatal injury to a fetus who is then born and lives with severe disabilities is an unambiguous wrong. Yet a fatal injury to the same fetus, resulting in early spontaneous abortion, may be a wrong but is not unambiguously or universally regarded as a wrong.

Fourth, most observers regard the maturing fetus as having an evolving moral status. That is, the propensity to regard the fetus as deserving protection and being under the care of the moral community grows stronger as the fetus approaches term. The Supreme Court's major abortion decision, *Roe* v. *Wade*, implies that by the third trimester the welfare of the fetus is an appropriate concern of the State, whereas in the first trimester no such grounds for State involvement exist.

Workplace fetal protection policies often further complicate these ethical problems. Because employers can be sued when exposure to workplace toxicants results in a damaged child, they institute such policies, which may impinge on the woman's employment opportunities.

MORAL PRINCIPLES AT STAKE

Three ethical principles are relevant to the issue of reproductive hazards in the workplace: 1) respect for persons, 2) beneficence, and 3) justice.[2]

Respect for Persons

The principle of respect for persons must be considered in the context of reproductive health hazards. This principle applies to both the *workers* exposed to hazards, and the *offspring* and *potential offspring* of these workers.

Workers

Respecting the autonomy of workers generally entails allowing workers to make their own informed and voluntary choices. This implies a duty on the part of those in a position to inform workers—principally employers, their labor unions, and the government—to disclose existing information about reproductive hazards in the workplace. Certain practical questions then arise: Should every conceivable risk be disclosed, no matter how poorly established or improbable? Is it sufficient to give the information to workers, or is there a duty to see that the information is understood?

Both of these questions have been asked about disclosure of risks in research and in medical treatment. One widely accepted answer to the first is to use a "reasonable person" standard: disclose those risks that a reasonable person would want to know about. This leaves out very improbable risks, although serious but not well-documented risks might have to be disclosed under the "reasonable person" standard. Reasonableness is again crucial in the answer to the second question: a reasonable effort must be made to ensure that the person has understood the risk. This includes eliciting what the person thinks he or she has been told, and attempting to correct any misunderstandings therein revealed. A second possible implication of properly informing workers might be a duty to seek out information that is likely to be relevant to a reasonable person's decision to accept hazardous employment. For example, it might literally be true for an employer to say that there is "no scientific evidence" that a certain chemical is hazardous to reproductive capacity in humans. But such a disclaimer is disingenuous if research has not been done on the question. It would also leave open the possibility that the substance has been found to be harmful in animals, or that suspicious cases have been found in humans, but that no relationship has been firmly established.

Whether employers or the government have an ethical duty to seek out information is not well established. Nor is the scope of such a duty well defined. Should it apply to all new chemicals, or only to those for which there is some strong reason to suspect that they pose a hazard to human reproduction—e.g., that a chemical is structurally very similar to a chemical known to be a reproductive hazard? The "duty to seek out information" is a plausible extension of the duty to inform.

Honoring the principle of respect for persons requires not merely informing workers, but also ensuring that their choices may be made voluntarily and without coercion. In reality, decisions about employment are seldom made on a purely voluntary basis and without financial or other possibly coercive pressures. People need to work for both financial and psychological reasons. Appreciating the ever-present constraints on choice with respect to employment does not vitiate the duty to foster voluntary choice, but does sometimes compel a settlement for less than absolutely free choice, and it focuses attention on the factors that make a choice more or less voluntary.

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[2] The section that follows is drawn from a contractor report prepared for OTA by Thomas Murray of the University of Texas at Galveston.

A multitude of factors can influence the "voluntariness" of a choice to accept hazardous employment. Individual alternatives are affected by: national and local unemployment rates and local employment options (whether, for example, the worker lives in a large city with many different industries, or a small, isolated one-company town). Other factors include the worker's qualifications, his or her financial status and needs, and the disincentives for seeking work elsewhere (e.g., the emotional and financial costs of moving to another community, or rules governing pension-vesting with a current employer).

Respect for persons in this context, then, involves informing workers about hazards, and attending carefully to the voluntariness of the choices they have in practice, not merely in principle. It could, for example, lead to a preference for a regulatory policy that provided alternatives to workers over one that prescribed an all-or-none choice. Reassignment with rate and seniority retention would be preferable, under this principle, to demotion or firing of workers.

Workers' Offspring

The principle of respect for persons offers little guidance about duties towards workers' offspring and potential offspring. The difficulty lies in the fact that embryo/fetuses, infants, and even children legally are not rational, "autonomous" beings, although they are potentially so. The Belmont Report of the National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research states that under the principle of respect for persons, "persons with diminished autonomy are entitled to protection."

As a moral principle, respect for persons has its clearest application in cases where the free, informed choice of rational adults is in jeopardy. In general, the interests of fetuses, infants, and children fall more naturally under the principle of beneficence, to be discussed next. In the context of respect for persons, the most that can be said about fetuses is that if they are to be brought to term, their capacity for autonomous thought and action in later life should not be impaired.

Beneficence

The principle of beneficence requires avoiding harm to others (sometimes referred to as "nonmaleficence") and maximizing the balance of benefits over harms. Beneficence comes into play in at least three relationships in the workplace: employers' duty to workers, workers' duty to offspring, and employers' duty to offspring.

Employers' Duty to Workers

Do employers have any duty to act beneficently towards employees? Both the specific and the general legal duties imposed on employers under the Occupational Safety and Health Act strongly imply that there is a corresponding ethical duty to avoid exposing workers to unreasonable risk of harm. It seems reasonable to view the OSH Act as the statutory codification of a growing social conviction that the duty exists on the moral level. If a moral duty exists, what is its scope? Does it extend beyond nonmaleficence, avoiding harm, to beneficience, maximizing the balance of benefits over harms?

There are limits to the duty to act beneficently towards employees. For example, the duty to act beneficently probably does not require employers to look after all of an employee's health risks, only those imposed or perhaps aggravated by conditions at work. Employers may choose to concern themselves with workers' health more broadly, for example, through health promotion programs designed to control smoking, alcohol consumption, or drug abuse, and some would argue that employers are required to do so. Beneficence, however, cannot require what cannot be done. Complete elimination of all risks is not ethically required when it is not practically possible. Therefore, employers may not have a duty of beneficence to workers, in the broad sense of a responsibility to maximize the balance of benefits over harms.

But even with these limitations, the principle of beneficence in the sense of nonmaleficence clearly applies to the case of employers' duties to avoid harm to workers' reproductive capacities and outcomes. Workers are harmed physically when their reproductive capacities are adversely affected. They are harmed in other ways when a workplace exposure leads to the loss of an embryo/fetus, or to the birth of a damaged child. The child is harmed physically in this case, but the parent may be harmed physically, emotionally, and financially as well. That damage to one's child is a harm to the parent has been given legal confirmation through successful "wrongful birth" suits. It seems reasonable to conclude that employers have a duty, derived from the principle of beneficence, to avoid harm to workers through damaging their reproductive capacities or injuring their offspring. Establishing the scope of this duty is complicated by uncertainties about the probability and magnitude of risks.

Workers' Duty to Their Offspring

Do parents have any duties to their offspring during the period of fetal development? What scope might such duties have? What are the implications of these duties, if they exist, for the workplace context?

When there is the intention to bring an embryo/fetus to term, then parents may well have certain duties to that child-to-be even while it is a fetus. For example, a person who gives drugs to a pregnant woman, knowing that the drugs will almost certainly cause gross physical or neurological abnormalities that will persist throughout life, has harmed that fetus. Most people would agree that the individual has harmed the child as surely as if the injury had been done after the child's birth. If there is a duty to avoid harming a child, then there may be a corresponding duty to avoid harming the fetus that will develop into that child. Generally, because a parent has a duty not to do grievous and irreversible harm to his or her child, it may be reasonable to assume that the same parent has a similar duty not to harm the same child before birth. This duty can sometimes conflict with the pregnant worker's autonomy in that her efforts not to harm her expected child may be constrained by workplace rules that compromise her employment opportunities.

The situation is much less clear when there is an intention to abort the fetus. To say that parents have a duty not to harm a fetus when a fetus may in fact be electively aborted raises the larger question of the moral justification of abortion, which is beyond the scope of this report.

If, however, there is a duty to the wanted embryo/fetus, the boundaries of that duty may be set. At their upper bound, such duties might equal but could not exceed the duties owed to newborn infants. This is helpful to note because it reminds us that more familiar issues are pertinent to this case. In particular, it points up a limitation to the duties towards wanted fetuses: beneficence requires one to do what is best for a person, on balance. It is not a duty to avoid any and all possible harms to the fetus, when that same action might gain some benefits to the fetus and avoid other harms. Practically, a parent might continue to work under conditions of mild or low probability of harm to a fetus, if the benefits to the fetus outweighed the likelihood of harm. Benefits could reasonably include those things made possible by the income and employment benefits derived from working, such as adequate prenatal health care, or better housing, food, and clothing for the child. Establishing that working parents may have a duty of beneficence towards their offspring, even as wanted fetuses, does not automatically mean that they must surrender their jobs, at least not when the sum total of benefits to their children-to-be might be negative.

From the standpoint of the management of exposure to reproductive health hazards, the importance of this point is that a parent who chooses to continue working in a mildly hazardous workplace is not necessarily violating any duty of beneficence to his or her unborn child.

Employers' Duty to Workers' Offspring

Do employers have a moral duty of beneficence towards the not-yet-born children of their workers? Morally, everyone has a general duty not to harm others. This would apply as well to the moral duty of employers towards workers' offspring. The scope of this duty (e.g., the amount of sacrifice demanded) is affected by whether there is any special relationship between parties and the nature of that relationship.

Under the general duty clause and specific standards, the OSH Act creates a legal duty to protect reproductive health and procreative ability in that workers should not be exposed to "recognized hazards" (see chapter 7).

There is no such legal duty with regard to "unrecognized hazards," although a tort suit alleging harm to a child while in a mother's womb might succeed. Thus, there is no clear relationship between the parent's employer and the fetus. Whatever duty might exist would almost certainly be less strong than the employer's duty to the worker-parent or the worker-parent's duty to the fetus. Nevertheless, logically, a moral duty exists not to do gratuitous or easily avoidable harm to the fetus. A case can illustrate that point.

If, as an example, a particular workplace exposure were to be quite harmless to the workers, but cause severe birth defects in fetuses carried by the workers, avoiding harm to the fetus could be a morally significant reason to alter the exposure. Furthermore, if the danger to the fetus were known in advance, the employer probably would have violated a moral duty not to do avoidable harm to the fetus.

The scope of employers' duty to their workers' fetuses is difficult to determine because of considerations that point in opposite directions. On the one hand, the lack of a clear relationship between employer and fetus, in combination with ambiguities in the moral status of fetuses, suggests that the duty is more narrow and less broadly compelling than the employer's duty to workers. While the worker-parent's exposure is to some degree voluntary, the fact that the embryo/fetus has not "consented" to be exposed to hazards should not lead to the implementation of a higher standard of protection for the fetus than for the worker-parent. What this underscores is the interaction of the principles of respect for persons and beneficence: the duty to protect some persons from harm may be in conflict with the duty to permit other persons maximum latitude for free and informed choice. Although both kinds of duties are important, they may at times conflict.

Whatever the duty between employer and fetus may be, there are questions about how it may apply in practice. There is a familiar doctrine in bioethics, in family law, and in medical decisionmaking for children, the mentally ill, the retarded, and other noncompetent persons. Known as the "best interest" standard, the doctrine urges that whatever decision is made be in the person's best interest.

Unless there is compelling evidence to the contrary, the judgment of what is in the child's best interest is left to the parents. Most experts would agree that the parents are well situated to decide where the child's interest lies; further, they will be motivated by concern for the child. Finally, on balance, it is better for the autonomy of the family to be preserved than to have others constantly meddling in the family's most intimate decisions.

It would be ironic if, on the one hand, wide parental discretion in health care decisions for their infant or child were to be permitted, while on the other, parents were to be denied authority to make decisions affecting the health and well-being of unborn children by being denied the right to employment that might have some effect on the fetus's subsequent development and chances for a decent life. This does not mean that parents should have absolute sovereignty over the life and death of their children, or that they can inflict with impunity any lifelong harms on their yet-to-be-born child. Courts can and have regularly intervened when parents, even with deeply grounded religious motives, have chosen a course of action that endangers the life of their child. But society has set a fairly high threshold of intervention: anything posing less than a clear and highly probable threat to life or health will not trigger intervention.

This analysis suggests that decisionmakers should seek to preserve parental autonomy, unless there is clear evidence of a highly probable threat to the infant-to-be's health or life. Therefore, policies designed to protect fetuses should grant substantial leeway to both male and female worker-parents. Information and education campaigns or policies that provide for transfer without penalties are to be preferred over policies requiring involuntary transfer or job termination. It is highly unlikely that a worker-parent, informed of a grave risk to his or her yet-to-be-born child's health and given alternative employment without loss of wages or seniority, would nonetheless insist on remaining in the more dangerous workplace and thereby expose the embryo/fetus to serious hazard. In such a case, involuntary transfer or termination might be a morally defensible choice. But it is difficult to imagine a potential parent voluntarily risking his or her wanted child's health in this manner.

Justice

Justice as an ethical principle is relevant to the regulation of reproductive hazards in two ways: in the differential impact on male and female workers, and in the allocation of burdens.

Differential Impacts on Male and Female Workers

In its most basic formulation, the principle of justice requires that like cases be treated alike. Policies that have a much heavier negative impact on workers of one sex may not be just because sex, like race and age, is an immutable characteristic. At a minimum, policies with differential impact require justification, which can take the form of showing relevant differences—demonstrating that the cases are not alike in important respects.

In the case of exposure to reproductive health hazards in the workplace, "fetal protection policies" have typically targeted women, who are much more likely to be removed from or denied jobs on the grounds that reproductive hazards exist. The background of employment discrimination against women underscores the importance of scrutinizing policies that have a disproportionate impact on women. The history of discrimination increases the burden of proof for those who propose such policies. Unless it can be shown that such policies are based on relevant and important differences, they must be regarded as unjust. If, for example, a substance were shown to have an effect on both men's and women's reproductive capacities, there would be no grounds for selectively excluding women from jobs involving exposure to the substance. There is good reason to redesign the workplace or the production process so that neither men nor women are exposed to dangerous levels of the substance, but no reason to expose men alone.

If after reducing exposure to as low a level as technologically feasible, the low levels were shown to affect only one sex, that could be a relevant difference, and justice might not be violated by a policy affecting workers of that sex. This assumes that there is good scientific evidence that the presumably unaffected sex is in fact unaffected. If there are no competent and statistically powerful studies confirming that there are no effects on the other sex, then a relevant difference has not been shown and justice has not been satisfied. This is particularly relevant for the management of exposure to reproductive health hazards since much of the research has focused on women alone, and therefore effects in men are more likely to be uninvestigated than nonexistent.

Does the obvious fact that women are the ones who actually carry the fetus constitute a relevant difference? It may, but only subject to the reservations noted earlier in the discussion of beneficence and respect for persons. Respect for persons and beneficence support policies that inform workers fully about the reproductive hazards to which they may be exposed, and that permit considerable discretion to men and women alike in deciding whether to accept work that may pose a reproductive hazard.

Allocating Burdens

A major issue is who should bear the burden of uncertainty. Uncertainty is and will always be a major component in the management of exposure to reproductive health hazards. For most substances either very little is known, or limited evidence exists suggesting that the substance is a hazard. The principles regarding allocation of uncertainty parallel those operating once a hazard is recognized.

Once the existence of a hazard is established, the primary task of management is to limit exposure. From the viewpoint of justice, this entails allocating burdens among all affected parties, including employers, workers, and consumers. No single formulation of the principle of justice is universally accepted in the contemporary United States that can be unambiguously translated into decisions about the allocation of burdens. In general, these issues are best decided through full public debate and congressional disposition. However, there may be some useful clarification stemming from the most general formulation of justice—treat like cases alike—and a distinction between the two principal burdens to be allocated— financial burdens and health burdens. For the most part, serious impairment to a person's health is perceived as a greater harm to that person's interests than are financial burdens, particularly when financial burdens are spread over a large number of individuals, with little impact on each. If the impairment to health were mild, and the financial loss catastrophic, the financial loss could be judged more serious. But in the great majority of cases, especially where the health of individuals is weighed against financial burdens that will be widely spread among stockholders and consumers, justice in the United States would favor avoiding the catastrophic health burden on the few in favor of the relatively insignificant financial loss to the many. Harms to health are more likely to be irreversible than monetary loss. And health may be a more fundamental good than most other goods. Health is, in an important sense, a precondition of the pursuit of most of the other goods that make up the "good life."

Many employers have explicitly noted that their concern about the potential harm to the offspring of workers is motivated by fear of tort actions that might be brought against them on behalf of children allegedly harmed by parental exposure to workplace hazards. The effort to avoid financial harms that could follow the successful prosecution of such suits is best viewed as an effort by employers to protect themselves from avoidable economic burdens, and thus to place the economic burden of denied employment back on the workers, usually female workers.

At least four broad strategies[3] are possible for achieving the socially desirable goal of protecting workers and their offspring. Each by itself entails a very different distribution of the burden of reproductive health: 1) transform the workplace so that the reproductive health of both workers and potential offspring is protected to the extent feasible, 2) transfer male and female workers at appropriate stages of their reproductive cycles to jobs that will substantially reduce risk, 3) permit and/or compel male and female workers to work in settings defined as posing some risk, and 4) refuse to hire fertile women or discharge pregnant women from jobs that pose some risk to the health of a fetus.

The first strategy begins with the moral assumption that those who benefit from the labor of others bear the primary obligation for providing a workplace where risk of harm is reduced as much as is technologically feasible. Because employers have the financial capacity to absorb the costs associated with adopting protective policies, and because they have the capacity to shift these costs forward to consumers, this approach involves the broadest distribution of the burden of meeting the problem of the protection of reproductive health.

Should some level of reproductive risk remain, even under the best of circumstances, it may still be necessary to protect male and female workers from risk of reproductive harm at points in the reproductive cycle. Like the first approach, the strategy of job transfer would place on employers the primary financial burden of protecting reproductive health. If job transfer would entail rate and seniority retention, the employer would be assuming the full burden. To the extent that workers would be expected to take on less desirable jobs at lower pay, the burden of protecting reproductive health would be shouldered by both employee and employer. If patterns of promotion and seniority rights would be disrupted by the reproduction-related transfer of workers, other workers would be forced to bear part of the burden of such policies.

The third strategy would shift the burden of reproductive harm to workers by permitting them to assume the risks. Though tort suits might be available to compensate for negative reproductive outcomes, the personal burden and social consequences of workplace-induced toxicity for workers' reproductive capacity and procreative capability would not be avoided.

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[3] The section that follows is drawn from a contractor report prepared for OTA by Ron Bayer of the Hastings Center.

The fourth strategy, which is reflected in some fetal protection policies, shifts the burden of protecting society from developmental hazards entirely to female workers, who are forced to bear the consequences of job deprivation and reduced earning power. The risk to the embryo/fetus has been substantially reduced with this strategy. However, the burden for reproductive hazards is borne by male workers who will be continually exposed, and by female workers who will be "protected" only when they are pregnant or planning to become pregnant. The employer is not likely to bear the burden of potential tort liability.

Each strategy allocates the burden in a different way. The choice of a particular strategy or mix of strategies necessitates a realistic appraisal of how the burdens are to be allocated and what the ethical justifications are for allocating the burdens. In addition, this discussion focuses only on the rationale of allocation of burden; the principle of differential impact on particular groups is not discussed. Obviously, both of these principles as well as those of respect for persons and beneficence would apply in the resolution of these dilemmas.

Appendixes

Appendix A

Reproductive Dysfunction in the Population

Introduction

Analysis of incidence rates and trends in reproductive dysfunction in the general population, along with evidence of nonoccupational causal factors, is important to this report because:

1. such analysis provides background rates against which to identify unusual increases in incidence that may be related to occupational hazards within population subgroups;
2. correlation of background rates with demographic and other variables may yield clues to the etiology of certain reproductive disorders;
3. nonoccupational exposure to reproductive health hazards may have synergistic effects with factors existing in workplace settings; and
4. a better understanding of the etiology of reproductive dysfunction may allow identification of hazards and development of preventive or protective measures that could be applied in the workplace.

The following review is organized according to the two populations affected by reproductive hazards—adults and offspring. Recent trends and possible causes of infertility in both men and women are discussed, and major impairments manifested in the offspring—including infant mortality, low birth weight, and birth defects—are examined. Depending on available data, incidence rates are analyzed for correlation with demographic variables such as age, race, geographic region, and socioeconomic level.

Infertility

Incidence of Infertility

There have been three national surveys of reproductive capacity among married American couples, in 1965 (128), 1976 (163), and 1982 (117). Despite methodological differences between the first and the latter two surveys, some trends in infertility emerge from the data.

It is important to note that infertility is often a temporary phenomenon. Estimates of infertility may therefore vary widely, according to the specified interval of inability to conceive. The common medical definition of infertility is used here: inability to conceive after 1 or more years of marriage during which contraception is not used. This 1-year period appears reasonable since almost 90 percent of the infertile couples surveyed were infertile for 18 months or more, and almost half for 30 months or more (117).

In 1982, approximately 2.4 million married American couples, or 8.4 percent of those in which the wives were of childbearing age (15 to 44), were classified as "unintentionally" infertile. Of these couples, 70 percent wanted to have a child. Thus, in 1982, a total of 6 percent of married couples involving women of childbearing age who wanted to have a child were thwarted by infertility (117). The epidemiologic profile of infertile couples in the United States reveals a tendency for them to be black, the wife to be age 30 or over and have less than a high school education, and for the couple to have experienced one or no previous live births (117a).

In the 1965-82 period, the overall unintentional infertility rate for all women aged 15 to 44 appeared to drop from 11.2 to 8.4 percent (table A-1). However, the percentage of couples in which one or both partners had been surgically sterilized more than doubled in this period, from 16 to 39 percent (117), thus reducing the population at risk for infertility. If the infertility rate is calculated only for the at-risk population (i.e., excluding those who were surgically sterilized), overall infertility did not in fact change (table A-2). There was, however, a significant increase (3.6 to 10.6 percent) in infertility among the 20 to 24 age group of wives during this period (117). The still-sizeable gap in infertility between couples in which the wife is over 30 and those in which the wife is younger may therefore be narrowing.

Although the 1982 data have not yet been analyzed by racial subgroups, the 1965 and 1976 data reveal significant race-specific trends. Unintentional infertility increased by 350 percent among black couples in which the woman was 20 to 24, and 58 percent among those in which the woman was 25 to 29. Smaller but significant increases were found among white couples in which the women were in the same age group (117a). Blacks exhibited consistently higher overall infertility than whites: 55 percent higher in 1965, and 92 percent higher in 1976.

These racial differences may also be partly explained by trends in surgical sterilization: the percentage of couples seeking surgical sterilization is rising more rapidly among whites than blacks, reducing the num-

Table A-1.—Percent Distribution of Currently Married Women 15 to 44 Years of Age by Infertility Status: United States, 1965, 1976, and 1982[a]

	Number in thousands[b]				Infertility status (percent distribution)								
					Surgically sterile			Infertile			Fecund		
Age and parity	1965	1976	1982	Total	1965	1976	1982	1965	1976	1982	1965	1976	1982
Total	26,455	27,488	28,231	100.0	15.8	28.2	38.9	11.2	10.3	8.5	73.0	61.6	52.6
Age:													
15-19 years	1,032	1,043	612	100.0	0.6[c]	1.0[c]	0.3[c]	0.6[c]	2.1[c]	2.1[c]	98.9	96.6	97.7
20-24 years	4,397	4,977	4,130	100.0	3.1	4.5	8.2[c]	3.5[c]	6.4	9.7[c]	93.4	89.2	82.1
25-29 years	4,953	6,443	6,442	100.0	9.5	16.6	19.6	6.5	9.0	7.0[c]	84.0	74.4	73.4
30-34 years	5,074	5,736	6,482	100.0	17.0	36.2	43.6	11.6	10.3	7.7[c]	71.3	53.5	48.7
35-39 years	5,700	4,814	5,783	100.0	22.8	45.3	58.1	14.2	12.5	10.3	63.0	42.2	31.6
40-44 years	5,298	4,474	4,783	100.0	26.8	49.0	66.7	20.2	15.9	9.0[c]	52.9	35.2	24.3
Parity:													
0	—	5,235	5,098	100.0	7.3	5.6	9.9	14.5	18.1	19.6	78.2	76.3	70.5
1	—	5,571	5,891	100.0	7.5	8.8	17.7	17.2	12.4	10.8	75.3	78.8	71.7
2	—	7,638	9,042	100.0	14.2	32.3	46.9	9.3	6.0	5.0	76.6	61.7	48.1
3 or more	—	9,045	8,201	100.0	21.5	49.8	63.3	9.4	7.9	3.8	69.0	42.3	32.9

[a] Statistics are based on samples of the household population of the conterminous United States.
[b] Weighted numbers not available by parity for 1965
[c] Indicates that the statistic has a relative standard error of 0.3 or greater

SOURCE: W. D. Mosher, "Fecundity and Infertility in the United States, 1965-1982," NCHS, paper presented at the annual meeting of the Population Association of America, Minneapolis, MN, May 3-5, 1984.

Table A-2.—Percent of Women, Presently Married, 15 to 44 Years of Age, Who Were Infertile, Excluding Those Surgically Sterile, United States

	Percent infertile		
Age and parity	1965	1976	1982
15-44 years	13.3	14.3	13.9
15-19 years	0.6[a]	2.1[a]	2.1[a]
20-24 years	3.6[a]	6.7	10.6
25-29 years	7.2	10.8	8.7
30-34 years	14.0	16.1	13.6
35-39 years	18.4	22.8	24.6
40-44 years	27.7	31.1	27.2
All parities	13.3	14.3	13.9
0	15.6	19.2	21.8
1	18.6	13.6	13.1
2	10.8	8.9	9.3
3 or more	12.0	15.8	10.3[a]

[a] Indicates that the statistic has a relative standard error of 0.3 or greater.

SOURCE: W. D. Mosher, "Fecundity and Infertility in the United States, 1965-1982," NCHS, paper presented at the annual meeting of the Population Association of America, Minneapolis, MN, May 3-5, 1984.

ber of white couples at risk for unintentional infertility. Black couples seek surgical sterilization less often, perhaps because they are more likely to have already experienced infertility (117a). Many other factors, including economic and cultural differences, may contribute to this phenomenon.

The 1982 data were analyzed for differences in sexual activity that may cause a couple to be classified as infertile. No difference was found in the frequency of intercourse of infertile couples compared with those that did not report fertility problems (117).

A large proportion of couples classified as infertile at the time of these surveys were probably only temporarily affected and later recovered. In a longitudinal study of infertile couples, 38 percent eventually achieved pregnancy following a mean infertility duration of 3 years (21). Furthermore, a large percentage of these recoveries were treatment-independent: 35 percent of untreated couples recovered spontaneously, compared with 41 percent of treated couples. Added to the category classified as "treatment-independent" were 31 percent of the treated couples who became fertile more than 3 months after the last medical treatment or 12 months after therapeutic surgery.[1] In sum, "treatment-independent" pregnancies constituted 61 percent of all pregnancies that occurred in this study (21). In other words, most of the infertile couples who regained their reproductive capacity did so independently of medical treatment.

Spontaneous Abortion

Spontaneous abortion, defined here as embryonic or fetal death before the 20th week,[2] is usually not included in the medical definition of infertility. However, since the inability to bring a pregnancy to term causes virtual infertility, spontaneous abortion is discussed here. Spontaneous abortion may also indicate fetotoxicity and/or severe physiological or genetic defects; in about 50 percent of first trimester spontaneous abortions, the fetus exhibits a chromosomal abnormality (50). One study of couples with a history of 3 or more consecutive spontaneous abortions identified possible causative abnormalities in 56 percent of the couples. The most common defects were anomalies of the uterus (15 percent), infections of the uterine lining (15 percent), and cervical incompetence (13 percent). Hormonal dysfunction was identified in 5 percent and chromosomal abnormalities in 3 percent of the couples (145). Several additional factors associated with birth defects (discussion follows) are also thought to increase the risk of spontaneous abortion.

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[1] The specified intervals were later criticized as being too short to be certain that subsequent pregnancies were treatment-independent (43,93).

[2] Fetal deaths beyond 20 weeks are included in perinatal mortality statistics and are considered obstetrical rather than fertility effects (67).

The incidence of spontaneous abortion is difficult to determine, largely because most occur during the first few weeks after conception when the woman may not yet know she is pregnant and embryo/fetal loss is difficult to detect. Estimates of spontaneous abortion range from 30 to 75 percent of all pregnancies (1). One prospective study using very sensitive pregnancy indicators found that 43 percent of the conceptuses were lost by the 20th week of pregnancy. Only one-fifth of these losses (9 percent of the total) were clinically apparent (110).

While retrospective abortion data are often more accessible than medical records or prospective data, there may be limits to the usefulness of recalled abortion data. Failure to recall an abortion may be related to:

- the time elapsed since the event,
- the total number of births or spontaneous abortions experienced by the woman,
- a woman's age at the time of pregnancy,
- the gestational age of the fetus at the time of abortion,
- medical treatment and hospital admission, and
- social class and education.

A 1984 study of the accuracy of spontaneous abortion recall found that one of every four recorded spontaneous abortions was not later recalled (179).

The likelihood of spontaneous abortion increases with maternal age, especially after age 40, and is greater among those who have previously had spontaneous abortions (124). One estimate for clinically apparent spontaneous abortion among first pregnancies in women under age 30 ranges from 8 to 11 percent; in women over age 35, estimates range from 15 to 22 percent (67).

Causes of Infertility

Infertility is attributed in roughly equal proportions to husbands and wives among married infertile couples (54). The etiology of a given reproductive disorder may differ from situation to situation, and is often difficult to pinpoint: estimates of the percentage of infertility cases for which no cause can be pinpointed ranges from 6 (168) to 50 percent (81).

The multiplicity of factors that frequently contribute to a given case of infertility can further obscure the etiology and thus hinder effective treatment: in one study, 40 percent of 141 extensively examined infertile couples were found to have multiple factors contributing to their reproductive impairment—7 percent had 3 or more identified contributing factors (168). In addition, individuals may be affected differently by a given agent due to differences in characteristics such as age, health, and personal habits that can affect the risk of reproductive damage.

A separate in-depth study of about 500 infertile couples found that the three major primary causes of infertility were ovulation defects, tubal disease, and "male factor" problems, including sperm count below 10 to 20 million sperm per milliliter (million/ml) of semen, impaired sperm motility, or psychiatric problems leading to impotence or loss of libido. Approximately 20 percent of diagnosed cases were attributable to each of these disorders, which therefore collectively caused infertility in 60 percent of diagnosed couples. No diagnosis was possible for 25 percent of the couples (77). Among major secondary causal factors were unreceptive female mucus (severe infection or immune response to sperm causing agglutination), endometriosis (unusual location, proliferation, and dispersal of tissue resembling that of the uterine lining), cervical abnormalities, and luteal phase deficiency (insufficient secretion of hormones necessary to maintain the uterine lining). In this study, 52 percent of the infertile couples eventually bore a viable infant; 90 percent conceived within 1 year of initial medical consultation (77).

The proportion of fertile[3] couples in the United States fell from 73 to 53 percent between 1965 and 1982. This drop is paralleled by a twofold increase—from 16 to 39 percent—in the percentage of couples in which one or both partners have been surgically sterilized by tubal ligation, vasectomy, or hysterectomy (117). While this trend in surgical sterility is not further explored here, it is useful for isolating, and sometimes explaining, trends in unintentional infertility.

The following two sections discuss the rates and possible causes of specific conditions contributing to reproductive impairment among women and men. Depending on available information, the following categories of causal factors are discussed for both women and men: 1) **environmental factors,** including pollutants; 2) **pathological factors,** including infectious disease; 3) **heritable factors,** such as inherited syndromes; 4) **iatrogenic factors** (i.e., side effects of medical treatment), including contraception and therapeutic drugs; 5) **nutritional factors**; and 6) **sociobehavioral factors,** including "recreational" drugs, stress, exercise, and paternal or maternal age.

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[3] Couples defined here as fertile encompass all those capable of conceiving, including those who are using contraception.

Women.—The six causal factors listed may contribute to the following female reproductive impairments, all of which can contribute to infertility: amenorrhea (absence of menstrual cycle), oligomenorrhea (reduced frequency of menstrual cycles), anovulatory cycles (a cycle during which no egg is released), premature menopause (cessation of menstrual cycling before the mid to late 40s) and spontaneous abortion.

Environmental Factors.—Two trends in exposure to environmental reproductive hazards may contribute to the increase in infertility among younger couples: 1) the increasing proportion of women entering the work force and thus potentially being exposed to reproductive health hazards in the workplace (6); and 2) the possible increased exposure of couples to environmental toxins that impair fertility (12).

Among the many environmental pollutants that may impair fertility among women are DDT analogs and polycyclic aromatic hydrocarbons. The banned pesticide DDT and the DDT analogs currently in use, such as methoxychlor, mimic the action of the sex hormone estrogen. Laboratory animals exposed to these pesticides exhibit impaired fertility and disturbances in mating behavior (57,90,171) (see chapter 4). Polycyclic aromatic hydrocarbons are common environmental pollutants produced by the burning of fossil fuels (e.g., oil, gasoline) and cigarettes, and the production of synthetic fuels (98,129). In experiments with laboratory animals, these hydrocarbons have been shown to destroy developing egg cells (37,101,102), produce ovarian tumors (70), and decrease fertility (91,100).

Pathological Factors.—Both the rise in infertility among younger couples and the concurrent decrease in infertility among older couples have been linked to trends in sexually transmitted disease. The rise in infertility among young couples may be partially due to the rising incidence through the 1960s and early 1970s of gonorrhea among young women (153). Gonorrhea can cause pelvic inflammatory disease (PID) with accompanying infertility (174). Reported cases of gonorrhea tripled between 1965 and 1975 (24) with the highest rates evident for women aged 20 to 24, who have also shown the largest increases in infertility (157). Since 1975, the number of reported gonorrhea cases has exhibited a slow decline. Even with declining morbidity, persons aged 20 to 24 continued to account for 30 to 40 percent, and persons 15 to 19 for nearly 25 percent of all reported cases of gonorrhea each year. By 1982, rates for women 15 to 19 exceeded those for women aged 20 to 24 (153).

Sexually transmitted infection by the chlamydia organism is a sometimes-unrecognized cause of infertility, although it has been described as epidemic in the United States (63). A study of pregnant women attending a prenatal clinic reported chlamydial infections in 5 to 10 percent of the women, a rate up to 10 times higher than that for gonorrhea, which was found in only 1 percent of the patients (63). Like gonorrhea, chlamydial infections can cause PID. In fact, chlamydial PID may be more likely to cause infertility than gonorrhea-induced PID (63). The high rate of "silent" chlamydial infections that are not clinically apparent, the lack of national incidence data, and frequent misdiagnosis have caused this disease to be both underestimated and undertreated.

From 1975 to 1981, the hospitalization rate for PID rose slightly overall for women aged 15 to 44 (174). High-risk groups included women in their 20s, divorced or separated women (1.7 times more likely to be hospitalized than single or married women), and nonwhite women (whose hospitalization rate was 2.5 times that of white women). Other factors that may have contributed to the recent rise in PID include the early age at which the baby-boom generation became sexually active, the tendency to have intercourse with a greater number of partners (187), and the shift away from contraceptives, like the condom, that protect against PID (117a,148). It has been projected that if these rises in venereal and pelvic inflammatory diseases continue among young women, 11 percent of women born in 1955 will become involuntarily sterile as a result of these diseases alone (24).

This rise in PID has also been linked to a near threefold increase in the incidence of ectopic pregnancy, from 4.8 per 1,000 live births in 1970 to 14.5 per 1,000 live births in 1980 (155). Ectopic pregnancy consists of implantation of the fertilized egg outside the uterus, often in the fallopian tubes that normally guide the egg from the ovary to the uterus. PID causes about 50 percent of ectopic pregnancies (63). Ectopic pregnancies must virtually always be terminated to save the mother's life and can cause temporary infertility. The infertility can be permanent if there is irreparable damage to the fallopian tubes. In one study of women with recent removal of an ectopic pregnancy, only about 20 percent eventually achieved live birth and only after at least 1 year of infertility (126).

Heritable Factors.—Maternal sickle cell anemia has been associated with increased risk of spontaneous abortion (115). Myotonic dystrophy, an inherited degenerative neuromuscular disease, can cause amenorrhea, although afflicted females in the younger age groups sometimes manage to conceive (133). Several inborn hormonal imbalances, when untreated, can compromise fertility in females. For example, imbalanced synthesis of steroids by the adrenal gland (congenital adrenal hyperplasia) is the most common cause of ambiguous external genitalia at birth. Without hormone replacement therapy, this disorder can result in infertility among these females (132). Untreated gonadotropin deficiency can result in amenorrhea and the failure of female secondary sex characteristics to appear. Untreated hypothyroidism has been associated with increased stillbirth rates and decreased fertility (66).

Some gross chromosomal abnormalities have also been linked to female infertility. Females born with only one of the normal pair of X chromosomes exhibit impaired ovarian function and are infertile (135). Women born with an extra X chromosome sometimes undergo premature menopause (139; for an in-depth review of heritable disorders that adversely affect female fertility, see ref. 135).

Iatrogenic Factors.—The use of oral contraceptives, which has rapidly increased since their release to the public in 1960 (166), can cause temporary infertility. Women may take longer to conceive after discontinuing use of oral contraceptives than those who use other or no methods of contraception (88). Two recent studies have linked IUD use to pelvic inflamatory disease and primary tubal infertility (22,25). Relative risk of developing primary tubal infertility differs depending on type of IUD used.

Both radiation and chemotherapy treatments for cancer can cause reproductive impairment in women. The effects of both treatments are age- and dose-related (20); both can cause ovarian damage and impaired fertility, to which prepubertal girls are more resistant than older females. Abnormal ovarian function can surface as irregular, anovulatory, or absent menstrual cycles or abnormal levels of certain sex hormones. The risk of complete ovarian failure due to chemotherapeutic drugs increases after the late 20s with clinical symptoms, including amenorrhea and low postmenopausal-like levels of certain sex hormones, that produce insomnia, irritability, and depressed libido (20). Furthermore, use of combinations of such drugs seems to increase the risk of ovarian failure.

Finally, most treatments for infertility (including many drugs that induce ovulation and some surgery) are associated with increased risks of spontaneous abortion among ensuing pregnancies. This effect may be a direct result of the treatment or due to an incomplete cure of the original reproductive impairment (67).

Nutritional Factors.—Changes in diet and body composition, many of which are associated with exercise, can affect female reproductive function. Substantial weight loss, low body weight-for-height, low percentage of body fat (40,41,42), and high energy consumption (173) have all been implicated in amenorrhea. The higher prevalence of menstrual disorders reported among college women compared with the general population has been associated with weight loss of 20 pounds or more—11.3 percent of college women reported oligomenorrhea and 2.6 percent reported amenorrhea (9) compared with 10 percent and less than 1 percent, respectively, in the general population (150). The severity of menstrual irregularity among those college women tested was associated with older age at menarche (9).

Malnutrition and starvation, including that induced by anorexia nervosa, can also delay menarche in young girls, who may then be permanently sterile. Malnutrition and starvation can also result in amenorrhea in older women (10).

Sociobehavioral Factors.—The recent tendency to delay childbearing to later years (161), when infertility is comparatively high, may result in an overall rise in infertility, especially in the older age groups (6). The reason for this age-related rise in infertility is unknown but may be partially due to the cumulative impact of infectious, occupational, and environmental agents as well as endogenous changes with age. The latter, "normal" age-related changes, could include changes in neuroendocrine hormone function, ovarian dysfunction, and increased incidence of spontaneous abortion.

Although many so-called recreational drugs, both licit and illicit, are suspected to have detrimental effects on reproductive ability, chronic cigarette smoking and heavy alcohol consumption have been most strongly implicated. Cigarette smoke can cause a dose-related lowering of the age of menopause, a sign of oocyte depletion (99). Smoking also increases the risk of spontaneous abortion to almost twice that of nonsmokers, according to one study (79). Women who drink the equivalent of one or more ounces of absolute alcohol twice a week (or at least four drinks per week) are almost twice as likely to abort spontaneously than women who drink less (78).

Other widely used licit and illicit drugs have been more tentatively linked to fertility problems. Barbiturates, some tranquilizers, marijuana, and narcotics may affect neural input into the hypothalamus, the brain center that orchestrates many of the body's endocrine functions, including the secretion of sex hormones. Marijuana can alter patterns of sex hormone secretion and inhibit ovulation in lab animals (138). Narcotics, such as heroin, may impair libido, potency, menstrual regularity and fertility, and may increase the incidence of spontaneous abortion (176).

Women who exercise strenuously, such as marathoners and ballet dancers, may exhibit some reproductive dysfunction, including delayed menarche, amenorrhea, and an abbreviated, dysfunctional luteal phase of the menstrual cycle. The reasons for these effects are largely unknown. However, exercise-induced decrements in reproductive function can usually be reversed through changes in lifestyle (23,143).

Men.—Major male reproductive dysfunction is associated with abnormalities in semen or sperm, ejaculatory failure, and impotence. The three characteristics of semen that are most strongly associated with infertility are low sperm count, reduced sperm motility, and abnormal sperm morphology—all of which may be interrelated (14,96). Most infertility clinics base their evaluations on all three of these parameters (see chapter 5). Standards of normalcy for these characteristics, however, as well as the degree to which the semen must deviate in order to cause infertility, remain controversial (86,105).

Although sperm count is commonly used as a measure of male fertility, neither a distribution of sperm counts among normal men nor a "normal" sperm count have been established. Sperm counts vary considerably between men and even within the same man according to age, sexual activity, season of the year, general health, and quantitation technique used (54). For example, daily sperm production declines significantly with age (68)—the number of sperm per ejaculation has been reported to decline by 30 percent in the 60 to 70 age group and by a further 20 percent in men 70 to 80 (48). In general, however, sperm counts below 10 million/ml are thought of as low and are correlated with increased risk of infertility (105,188). Yet people with "low" sperm counts are not always infertile. For example, one clinical study reported that 50 percent of men with sperm counts below 10 million/ml were able to initiate a pregnancy (2).

Sperm motility may also decline with age, especially after age 40 (92). Antisperm antibodies in the semen or in the cervical mucus of the female are associated with sperm agglutination, impaired motility, and infertility (96). Such antibodies can impede sperm penetration of the female's cervical mucus and egg (3). Antisperm antibodies are often found in the blood of infertile couples (53). One study reported that in more than 30 percent of infertile couples tested, at least one partner possessed antisperm antibodies in the blood, and that these couples had a lower incidence of subsequent pregnancy (108) (see chapter 5 for further discussion of seminal abnormalities as indices of reproductive impairment).

An 8-year study of donor semen at one infertility clinic reported a decline in semen quality, including lower sperm counts and more morphological abnormalities. Sperm motility remained constant. If this trend continues, and increasing numbers of donors have to be rejected, in 5 or 6 years none of the prospective semen donors will meet minimal standards (85). The significance of and reason for this decline are unknown.

Environmental Factors.—Analogs of DDT and certain other pesticides, some of which are still in use, mimic the action of estrogen and may affect male fertility (see chapter 4). Whether background levels of pesticides in the environment affect male reproductive function is unknown. (A comprehensive review of chemicals affecting human sperm appears in (184).)

Pathological Factors.—Several infectious diseases can cause inflammation of the testes and subsequent failure of spermatogenesis. These diseases include mumps, tuberculosis, gonorrhea, syphilis, typhoid, influenza, and smallpox (52). Testicular failure, accompanied by androgen deficiency, may also occur as a result of hypothalamic or pituitary disease. Estimates of the incidence of primary (i.e., testicular) endocrine defects among subfertile men range from 0.5 to 3 percent (26,167,169). Most infertile men have hormonal imbalances that are attributable to other infertility-causing pathological conditions (183).

Varicocele, or abnormal dilatation of the veins leading from the testes, may contribute to male infertility. Because varicocele is correctable by surgery (149), this disorder has been the subject of much recent research as a possibly reversible cause of male infertility.

Infants who are born with both ovarian and testicular tissue (for reasons that are usually unknown) are known as "true hermaphrodites." Due to the characteristic testicular degeneration, hermaphrodites are usually infertile as males, but have occasionally been shown to be fertile as females (135).

Some major psychiatric illnesses, such as schizophrenia, manic-depression, depression, and anorexia nervosa, are associated with infertility (10). The problem is complicated by the fact that psychotropic drugs used to treat these disorders may also impair fertility. Stress may lower testosterone levels in men and may be associated with decreased sperm count (104).

Impotence can be caused by pathological factors such as injury or cancer of the spinal cord or brain, cardiovascular disease, and hormone imbalances (including diabetes), and is generally associated with debilitating illnesses (55).

Heritable Factors.—Several congenital disorders, including cystic fibrosis, are associated with infertility

(52) (table A-3). Diabetes, sickle cell disease, and renal failure may also depress sperm production. Gonadotropin deficiency can cause loss of libido, impotence, infertility, and lack of sexual development. Another inborn hormonal disorder involving abnormally high levels of prolactin (a hormone secreted by the pituitary gland) typically causes impotence, but there is no general agreement about its effect on spermatogenesis (183). Myotonic dystrophy, an inherited degenerative neuromuscular disease, is associated with testicular atrophy accompanied by decreased libido, impotence, and infertility in afflicted males (133). Certain congenital urogenital or neurological abnormalities can cause retrograde ejaculation, the most common form of ejaculatory failure, in which the semen passes backwards into the bladder. However, this condition is more commonly acquired than inherited, whether as a side effect of surgery or trauma to the urogenital region (55) or of neurological disorders.

Klinefelter's Syndrome, whose frequency among males is estimated at 0.1 percent (19), is caused by possession of an extra X chromosome and usually results in azoospermia (absence of sperm). The testes of males with this disorder are typically small and devoid of germ cells.

Iatrogenic Factors.—Radiation and chemotherapeutic treatments for cancer can cause germ cell abnormalities and destruction in human males. Common side-effects are azoospermia, oligospermia (low sperm count), impaired sperm motility, abnormal serum levels of certain sex hormones, and decreased sperm synthesis (11,20). Permanent sterility is a common side effect of chemotherapeutic drugs, although some degree of potency and libido is usually retained. Most men experience decreased libido during combination chemotherapy and one-third experience impaired potency and decreased sexual pleasure after such therapy ends (20,178). These side effects are generally less severe in prepubertal boys than in men. Clinical knowledge of the extent, synergism, and variability of gonadal toxicity for many of these chemotherapeutic treatments remains inadequate, however.

Table A-3.—Conditions That May be Associated With Azoospermia. Some of These Factors May Cause Reversible Azoospermia

- Genetic disorders:
  Klinefelter's syndrome
- Congenital disorders:
  cystic fibrosis
  myotonic dystrophy
- Hormonal defects:
  hypothalamus
  pituitary gland
  adrenal gland
  thyroid
  testes
- Severe illness or malnutrition
- Infection:
  mumps
  smallpox
- Drug therapy:
  cytotoxic drugs
- Irradiation

SOURCE: Office of Technology Assessment.

Antihypertensives, anticonvulsants, and psychotropic drugs can all cause impotence. In addition, adrenergic-blocking agents and surgery to the prostate and bladder neck may cause retrograde ejaculation (55).

Nutritional Factors.—Some food additives may be reproductive hazards: estrogenic hormones fed to cattle to promote growth may be ingested by the human consumer in amounts large enough to affect fertility (52).

Sociobehavioral Factors.—Many commonly used recreational drugs (licit and illicit) affect male fertility. Chronic intensive use of marijuana, for example, is thought to inhibit normal secretion of gonadotropins, the pituitary hormones directing gonadal function (138), and cause significant reduction in sperm count and changes in sperm morphology (58). Alcohol depresses the synthesis and secretion of testosterone by the testes (69,106). Chronic alcohol consumption and alcoholic cirrhosis lead to multiple hormonal disturbances, growth of the male mammary glands, decreased size and functioning of the gonads, and sometimes impotence (55). Narcotics also cause impotence and decreased sex drive. Heroin addicts show depressed levels of certain sex hormones (76,107) along with decreased sperm count and motility (138).

Chronic use of anabolic steroid hormones, composed of testosterone and its synthetic derivatives, can cause the body to manufacture less of its own testosterone and result in testicular atrophy, decreased sperm count, and temporary infertility in males (189). Anabolic steroids have been employed to build up muscle mass by some athletes, particularly body builders and weight lifters, for years. More recently, the appeal of anabolic steroids has broadened to other groups, such as runners, swimmers, and cyclists.[4] Steroids appear to be the class of chemicals most consistently affecting sperm characteristics (184).

Chronic sniffing of paint or lacquer thinner can produce tissue abnormalities in the testes along with faulty or suppressed sperm production (146). Smoking can impair sperm production and increase the number of malformed sperm in the ejaculate (52).

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[4] Women, as well as men, may use anabolic steroids in sports training. Because their bodis produce only tiny amounts of testosterone, female athletes potentially will experience more of an effect on muscle mass from steroid use than will male atheletes (189).

Demand for Infertility Services

The proportion of all private physician visits devoted to infertility counseling rose by more than 30 percent between 1968 and 1980 (6). Several demographic and sociological factors may have contributed to this phenomenon:

- an increased number of infertile couples in the population,
- an increased proportion of infertile couples aware of and seeking infertility services,
- a growing number of physicians providing infertility services, and
- an increasingly pro-family social and political climate (6).

In 1976, about 6.9 percent of nonsterile,[5] married women aged 15 to 44 reported that they had used infertility services recently (i.e., consulted a doctor or other trained person within the previous 3 years) (164). Women aged 15 to 29 were significantly more likely to seek infertility services than those aged 30 to 44 (table A-4). Among childless women, blacks were nearly twice as likely to have recently used infertility services than whites. As might be expected, women of low parity[6] were also more likely to have had an infertility consultation than women of higher parity. The only statistically significant regional difference was found among white women: white women in the West were more likely to have recently sought services than those in the Northeast, North-Central, or South regions (164).

Table A-4.—Percent of Nonsterile Women (Currently Married, 15 to 44 Years of Age, United States, 1976) Who Had an Infertility Consultation in the Previous Three Years

Characteristic	All races[a]	Race	
		White	Black
All characteristics	6.9%	6.7%	7.4%
Age:			
15-29 years	8.5	8.4	8.8
30-44 years	5.0	4.7	5.9
Parity:			
0-1 parity	12.1	11.6	15.5
2 parity or more	2.6	2.6	2.3
Geographic region:			
West	8.6	8.8	3.4
Nonwest	6.5	6.2	7.9

[a] Includes white, black, and other races.

NOTE: Statistics are based on a sample of the household population of the conterminous United States.

SOURCE: Adapted from National Center for Health Statistics, "Use of Family Planning and Infertility: United States," *National Survey of Family Growth Vital and Health Statistics*, Series 23, No. 8, 1981.

Further study of trends in demand for infertility services and of the relevant demographic and sociopolitical variables may allow the health care delivery system to prepare for future demand, and to reach those subgroups expressing the greatest need.

Infant Mortality, Low Birth Weight, and Birth Defects

Background rates for infant mortality, low birth weight, and birth defects are important since many of the chemical, physical, and biological agents present in the workplace are suspected to adversely affect the gametes and/or fetus. A "baseline" rate is needed against which to compare abnormally high rates observed in certain settings and thereby pinpoint the responsible hazards. Causal factors and regional differences in these rates are identified where information is available.

Infant Mortality Rates

Death rates among both newborns (aged under 28 days) and infants (aged under 1 year) have dropped steadily since 1930 (see figure A-1). In 1982, 21 percent of all infant deaths were attributed to congenital anomalies (table A-5). This proportion has risen steadily in the 1900s (from 7 percent in 1916 to 18 percent in 1977) because the rate of congenital anomalies has dropped less rapidly than the overall infant death rate (figure A-2), and also because of improvements in pre- and post-natal care that have reduced the impact of other causes of infant death. Despite the recent decline in infant mortality in this country, however, the United States ranks 14th in an international comparison of infant death rates (94).

Almost 70 percent of infant deaths occur in neonates (i.e., within the first 28 days of life) (table A-5). More than half of infant deaths and three-fourths of neonatal deaths occur in low-birth-weight infants (162). The death rate remains substantially higher among black infants (figure A-3) and was almost twice that of white infants in 1982 (160). Black infants exhibit higher death rates for every major category in the National Center for Health Statistics data, except for cystic fibrosis (though there is overlap for specific defects within the congenital anomalies category). The rate among blacks is more than three times that of whites for deaths due to low birth weight or prematurity. This may be par-

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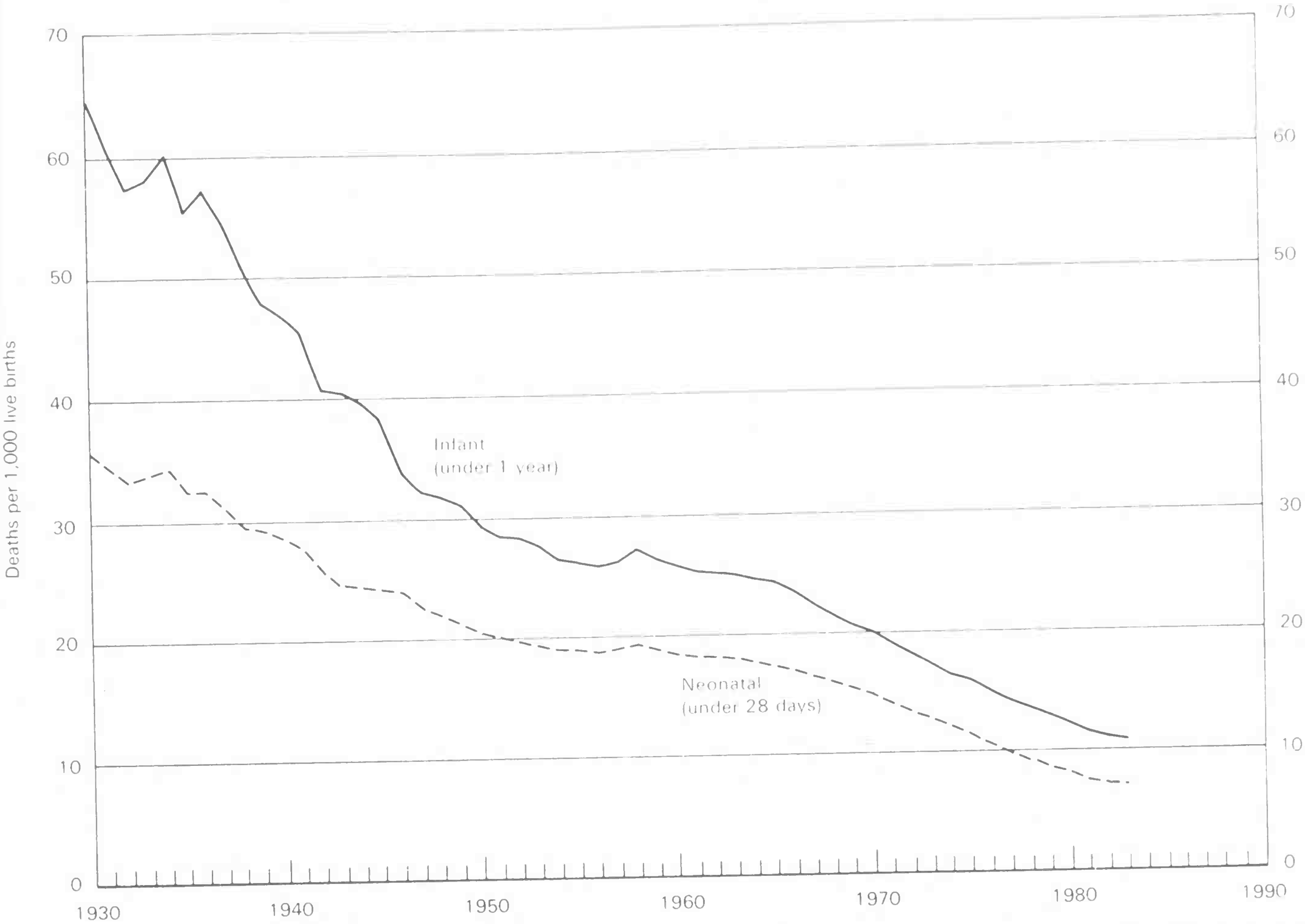
[5] Nonsterile is defined here as not being sterile due to contraceptive surgery, accident, or previous illness.

[6] Parity refers to the number of pregnancies a woman has carried to at least 20 weeks gestation (or 500-gram fetal weight).

Figure A-1.—Infant and Neonatal Mortality Rates, United States, 1930-83



SOURCE: National Center for Health Statistics, "Annual Summary of Births, Deaths, Marriages, and Divorces: United States, 1983," *Monthly Vital Statistics Report*, vol. 32, No. 13, Sept. 21, 1984.

tially due to the larger proportion of black mothers exhibiting maternal risk factors associated with bearing low-birth-weight infants.

Incidence of Low Birth Weight

Low birth weight, defined as 5 pounds 8 ounces (2,500 grams) or less, is strongly associated with both infant mortality and birth defects, including congenital malformations, mental retardation, and other physical and neurological impairments (159). Extremely low birth weight (4 pounds 7 ounces or less) is a leading cause of death among infants and a major factor in childhood disability (94). The percentage of newborns that are of low birth weight has declined in recent years, but less sharply than the neonatal death rate (figure A-4). In addition, the decline is due largely to the 21-percent drop in the incidence rate of **full-term** low birth-weight infants from 1970 to 1980, compared with only a 7-percent drop in the rate of **preterm** low birth-weight infants (74). These trends probably reflect improved neonatal care, but little improvement in the prevention of prematurity and fetal growth retardation (94).

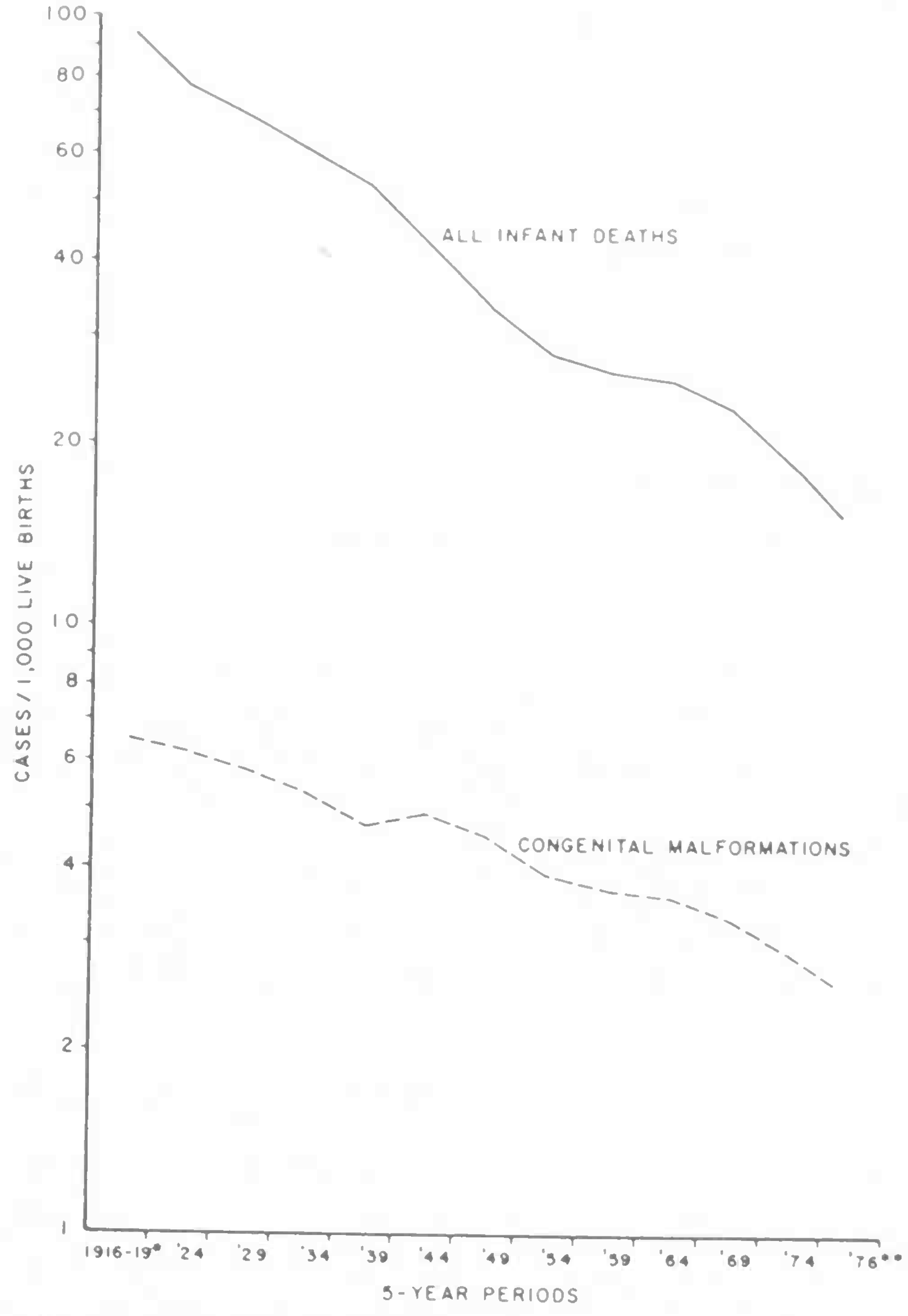
In 1981, 6.8 percent of all infants born were of low birth weight (159). The proportion of black infants of low birth weight was more than double that of white infants: 12.5 percent of black infants v. 5.7 percent of white infants. In a typical year with approximately 3.5 million births, the expected number of low birth-weight infants exceeds 200,000 (44). The racial difference in low birth weight has been attributed equally to racial differences in: 1) incidence of prematurity and 2) incidence of low birth-weight infants that are full- or post-term infants (there are few racial differences among preterm infants). In 1981, 17 percent of black infants were preterm compared with 7.9 percent of white infants. Among full- and post-term babies, the racial difference in low birth weight may be at least partially due to the larger proportion of all

Table A-5.—Infant Mortality Rates by Age and for Ten Selected Causes of Death, Based on a 10-Percent Sample of Deaths, United States

Age and cause of death	1982 (estimated) (rates per 100,000 live births)	Percent of total deaths
Total:		
Under 1 year	1,124.5	100.0
Under 28 days	762.4	67.8
28 days to 11 months	362.0	32.2
Congenital anomalies	237.0	21.1
Sudden infant death syndrome	127.2	11.3
Respiratory distress syndrome	107.2	9.5
Disorders relating to short gestation and low birth weight	98.8	8.8
Intrauterine hypoxia and birth asphyxia	39.7	3.5
Pneumonia and influenza	20.0	1.8
Birth trauma	15.4	1.4
Certain gastrointestinal diseases	7.3	0.6
Other conditions originating in the perinatal period	295.1	26.2
All other causes	177.1	15.7

SOURCE: Adapted from National Center for Health Statistics, "A Study of Infant Mortality From Linked Records by Birth Weight, Period of Gestation, and Other Variables: United States," *Vital and Health Statistics*, Series 20, No. 12, 1982.

Figure A-2.—Infant Mortality Rates in the United States, 1916-76



* = 4-year period; ** = 2-year period

SOURCE: G. P. Oakley, "Incidence and Epidemiology of Birth Defects," *Genetic Issues in Pediatric and Obstetric Practice*, M. M. Kaback (ed.) (Chicago: Year Book Medical Publishers, Inc., 1981).

black mothers who possess characteristics associated with bearing low birth-weight infants. These characteristics (159) include:

- being unmarried (81.4 percent of black mothers v. 18.2 percent of white mothers),
- having less than a high school education (35 percent of black mothers v. 20 percent of white mothers),
- prenatal care beginning after the second trimester or not at all (9 percent of black mothers v. 4 percent of white mothers), and
- multiple delivery (i.e., twins or triplets) (24.7 v. 18.8 per 1,000 live births for black and white mothers, respectively).

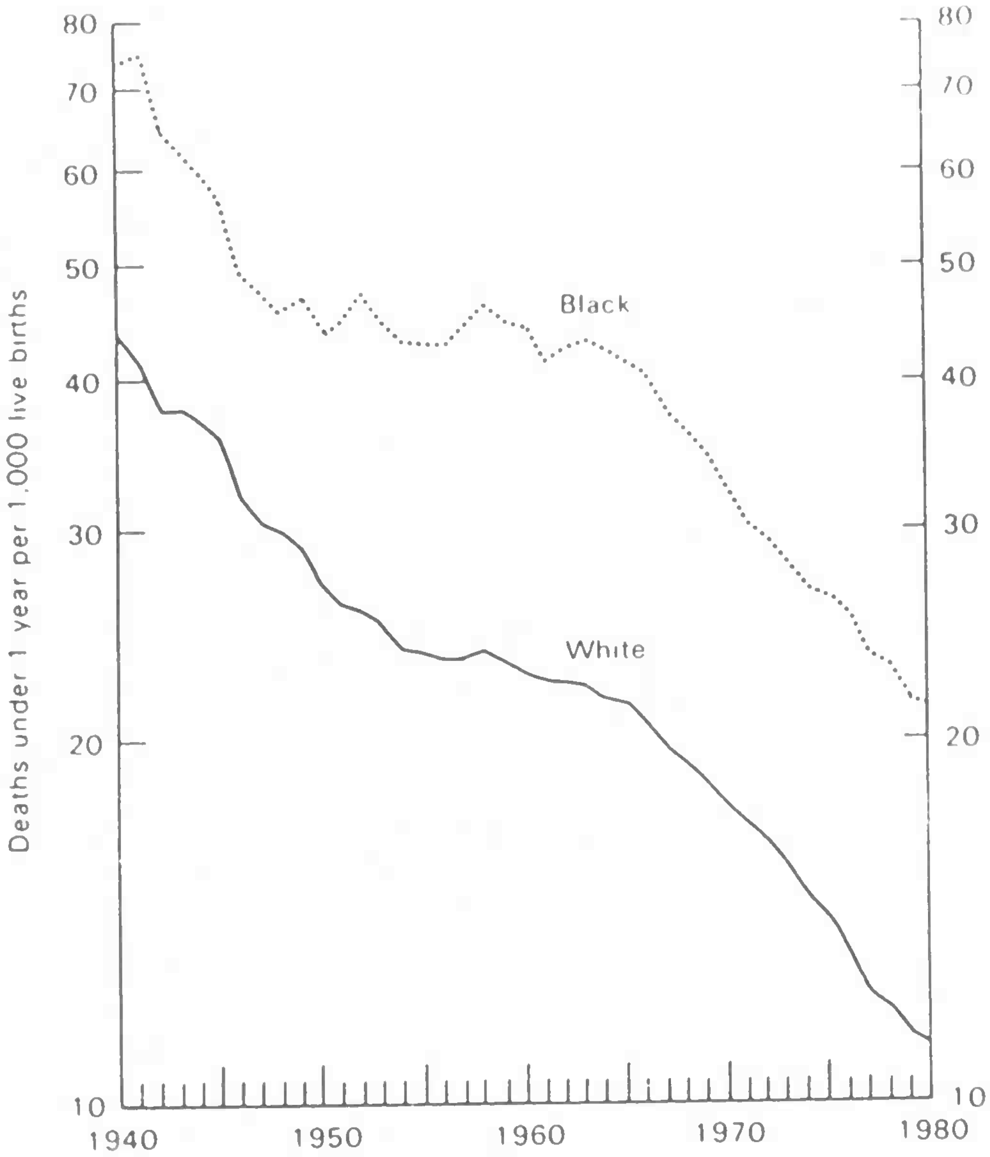
However, both the extent of prenatal care and the educational attainment of black mothers have improved in recent years (159).

In contrast, smoking and drinking during pregnancy—both of which are associated with low birth weight—are more prevalent among white women compared with both black and Hispanic women: 41 percent of white women drink during pregnancy compared with 24 percent of black women and 29 percent of Hispanic women; 26 percent of white women smoke during pregnancy compared with 22 percent of black women and 17 percent of Hispanic women; 12 percent of white women both smoke and drink during pregnancy compared to 9 percent of black women and 7 percent of Hispanic women (47,125). (Information on smoking and alcohol consumption during pregnancy appears in ref. 125.) Since the proportion of low birth-weight infants remains relatively low among white mothers despite these maternal risk factors, the adverse affects of smoking and drinking are either better-treated in white mothers, or compensated for by low prevalence of other maternal risk factors.

Women aged 25 to 34 years are the least likely to bear a low birth-weight infant: 5.8 percent of all births to this age class were of low birth weight. The percentages for those over age 40 and under age 15 are almost twice and three times as great, respectively (159).

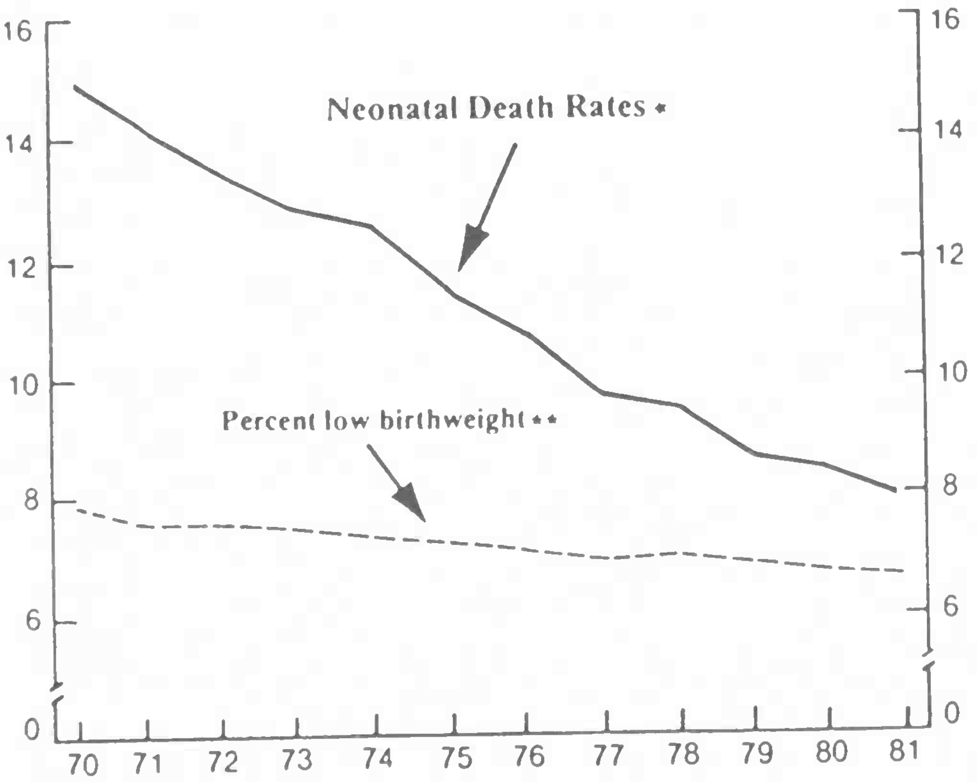
There is substantial variation from State to State in the percentage of low birth-weight newborns. The

Figure A-3.—Infant Mortality by Race, United States, 1940-80



SOURCE: National Center for Health Statistics, "Annual Summary of Births, Deaths, Marriages, and Divorces: United States, 1982," *Monthly Vital Statistics Report*, vol. 31, No. 13, 1983.

Figure A-4.—Neonatal Deaths and Low Birth Weight, United States, 1970-81



* Deaths under 28 days per 1,000 live births

** Percent of live births weighing 2,500 grams (5½ lbs.) or less

SOURCE: March of Dimes, *Facts/1984* (White Plains, NY: March of Dimes Birth Defects Foundation, 1984).

seven West North-Central States[7] have the lowest aggregate rate of low birth weight (5.8 per 100 total live births), while the nine South Atlantic States[8] have the highest rate (8.0 per 100 total live births). However, this regional variation may be due largely to the disproportionate number of black children born in those States with the highest low birth-weight rates. The five States with the highest rates of low weight births (District of Columbia, South Carolina, Mississippi, Louisiana, and Georgia) all reported substantially more black than white babies born in 1981. The regional variation observed **within** racial groups is largely reduced when the States with relatively few black births are excluded from the analysis (159).

Recent data demonstrate the strong nonrandomness of the geographical distribution of low birth weight in the United States. Clusters of low birth weight in the Rocky Mountain region and in certain Northern industrialized States suggest the strong influence of environmental factors such as altitude, mineral-extraction industries (e.g., lead, uranium, and silver mining), heavy industries (e.g., steel, automobile, and chemical), and agricultural spraying (44).

Birth Defects

A "birth defect" is defined here as any structural, functional, or biochemical abnormality, whether genetically determined or induced during gestation, that is not due to injuries suffered during birth. Birth defects afflict 1 of every 14 live-born infants (about 7 percent), or more than a quarter-million in the United States each year. Twice as many miscarriages and stillbirths occur annually, most of which are due to impaired fetal development (94). In 1982, 21 percent of all infant deaths were attributed to congenital anomalies, a proportion second only to that claimed by unspecified perinatal conditions (161) (table A-5). It is important to note that the problem of birth defects is not limited to infants: approximately 1.2 million people of all ages are hospitalized and 60,000 die as a result of birth defects each year (94).

Most of the birth-defects statistics used in this review are from the Birth Defects Monitoring Program (BDMP), which is based on the newborn discharge data (i.e., diagnosis at birth of both live- and still-born babies) of 955 participating hospitals nationwide (156). Although the BDMP data do not represent a random sample, the program remains the largest single source of uniformly collected and processed birth-defect data on newborn infants. Data from the NCHS include only live births (which lowers the reported incidence of commonly fatal conditions like anencephaly, absence of the brain), and are based solely on information gleaned from birth certificates. Birth certificates tend to underreport the more subtle birth defects not immediately apparent at birth. Therefore, this review employs NCHS data solely as a source for analyses of maternal data and other variables not considered by BDMP.

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[7] Minnesota, Iowa, Missouri, North Dakota, South Dakota, Nebraska, and Kansas.

[8] Delaware, Maryland, District of Columbia, Virginia, West Virginia, North Carolina, South Carolina, Georgia, and Florida.

It is important to note that analysis of temporal changes in incidence rates of birth defects is often limited by some degree of incomparability among data from different years. BDMP data are subject to changes in defect classification and are influenced by improvements in diagnostic abilities and public awareness that can elevate reported incidence.

Incidence of Selected Birth Defects.—For this discussion, 67 birth defects have been selected, according to incidence, severity of impact on those afflicted, and availability of data. The selected birth-defects data have been divided into 11 categories, according to the physiological consequence of the defect. Table A-6 lists these birth defects and their incidence in the United States in 1982.

Some of the most common defects involve the male urogenital system, including hydrocele (accumulation of fluid around the testes), hypospadias (opening of the urethra on the underside of the penis or on the pelvic floor) and undescended testicles (table A-6). Also relatively common are hip dislocation, patent ductus arteriosus (failure of the opening between the aorta and pulmonary artery to close after birth), clubfoot, and hemangioma (birthmark formed by blood vessels). Congenital metabolic disorders are the least common category overall, with incidences ranging from 0.1 to 0.6 per 10,000 total births. The other two rarest defects among those selected are congenital rubella syndrome and congenital glaucoma. Although the BDMP data do not provide an incidence figure for all chromosomal abnormalities combined, the aggregate rate has been estimated at 62 per 10,000 births (64).

Many of these birth defects have relatively low incidence rates, but the impact of their severe physiological effects on the families involved, and on society, is significant. Down syndrome, for example, has a relatively low incidence rate of 8 per 10,000 total births. Yet, because of the severity of its physiological and functional effects, it is the leading cause of major mental retardation in the United States (82). Similarly, neural tube defects (NTDs) such as spina bifida (incomplete closure of the spinal column) and anencephaly (absence of the brain) are relatively rare (table A-6), but have such devastating effects that their trends and etiology have been the subject of intense research.

The BDMP data are further subdivided into four regions of the United States: North East, North Central, South, and West (156) (figure A-5). Substantial variations are evident for only a handful of the selected birth defects. There are two major regional differences: one for NTDs and one for the North East region in general.

Three of the NTDs—anencephaly, spina bifida, and hydrocephalus (excess fluid in the brain)—exhibited incidences that are highest in the South (156). The reason for this regional trend is unknown. Data from BDMP and the Metropolitan Atlanta Congenital Defects Program (associated with the Centers for Disease Control) revealed that the incidence of "single" NTDs (those with no major associated defects) follows a decreasing East-West gradient and occurrs most frequently in white and in female newborns (75). Multiple NTDs do not fit this epidemiological profile. This indicates that there may be at least two distinct categories of causes for NTDs: one for single NTDs that is sex-race-region dependent, and another for multiple NTDs that is not dependent on these variables. Although several genetic and environmental mechanisms for the epidemiology of single NTDs have been postulated, they are not supported by conclusive evidence (75).

The second major regional difference extracted from the BDMP data is that, of the seven defects other than NTDs that exhibited substantial regional variation, six have much higher incidence rates in the North East compared with other regions: ventricular septal defect, Down syndrome, hip dislocation without CNS anomalies, rectal atresia and stenosis, clubfoot, and hypospadias (156). The rates are lowest for the first three birth defects in the South, and for the latter three in the West. Again, the reason for this regional variation is not clear, but valuable clues to the etiology of these defects may surface as more data are collected and more epidemiological correlations are made with environmental and genetic factors.

Causes of Birth Defects.—The causes of a given birth defect may vary from case to case, are often multiple, may involve synergistic effects such as the interaction of genetic and environmental factors, and are usually not known. Neural tube defects, for example, may be caused by a variety of chromosomal anomalies, maternal infections, genetic disorders, and teratogens (75). The causal role of chromosomal anomalies in NTDs has been questioned, however (66). Although the list of possible causal factors continues to grow, many birth defects are of unknown or, at best, speculative origin. Individuals may be affected differently by a given causal agent, and some may not be affected at all. Individual characteristics such as age, health,

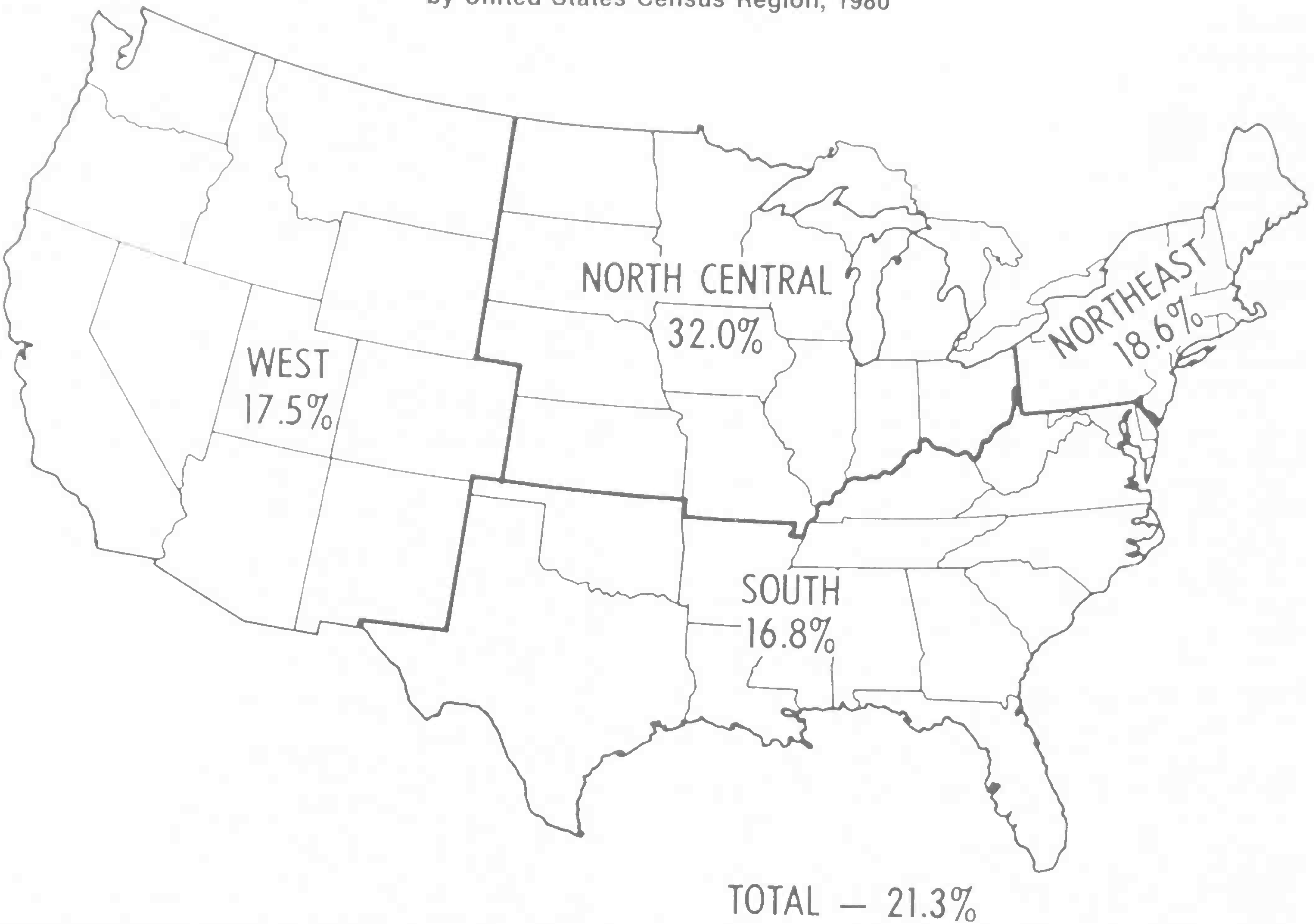
Table A-6.—Incidence Rates Per 10,000 Total Births of Selected Birth Defects, 1982

Condition	Incidence rate
Central nervous system:	
Total congenital anomalies of nervous system	18.4
Hydrocephalus (water on the brain)	5.6
Spina bifida (open spinal column)	4.9
Anencephaly (absent brain)	3.3
Microcephaly (small brain)	2.2
Encephalocele (hernia of brain)	1.0
Heart and circulatory system:	
Patent ductus arteriosus (failure of the opening between aorta and pulmonary artery to close at birth)	25.4
Ventricular septal defects (hole between lower chambers of heart)	14.7
Absence of umbilical artery	3.2
Valve defect, absence, or closure	2.2
Atrial septal defect (hole between upper chambers of heart)	1.5
Endocardial fibroelastosis (thickness of inner lining of heart)	1.2
Tetralogy of Fallot (a combination of congenital heart defects)	0.9
Transposition of great arteries	0.8
Other anomalies of the heart (not all-inclusive)	26.6
Urogenital system:	
Hydrocele (collection of fluid in membrane surrounding testes)	30.7
Undescended testicle	27.5
Hypospadias (urethral opening on underside of penis or on the pelvic floor)	27.0
Renal agenesis (absence of kidney)	1.6
Congenital ureteral obstruction	1.6
Cystic kidney disease	1.2
Indeterminate sex	0.6
Male genital anomaly N.E.C.[a]	5.3
Respiratory system:	
Agenesis (absence) of the lungs	2.5
Total anomalies of the nose	2.2
Anomalies of the larynx and/or trachea N.E.C.[a]	2.2
Congenital multi-system syndromes:	
Blood type ABO isoimmunization	144.8
Rh hemolytic disease in newborns	15.6
Down syndrome	7.9
Total monitored infections	4.2
Autosomal abnormalities (i.e., not on sex chromosomes) except Down syndrome	1.8
Total congenital syphilis	1.7
Fetal alcohol syndrome	1.2
Abnormalities of the sex chromosomes N.E.C.[a]	0.7
Congenital rubella	0.2
Congenital Metabolic Disorders:	
Total congenital amino acid disorders	0.6
Cystic fibrosis	0.6
Steroid metabolism disorder	0.2
Phenylketonuria	0.1
Total congenital carbohydrate metabolism disorders	0.1
Total congenital lipid metabolism disorders	0.1
Gastrointestinal tract:	
Total cleft lip and cleft palate	13.4
Intestinal anomalies N.E.C.[a]	4.9
Rectal defect, absence, or closure	3.0
Tracheal-esophageal fistula (opening between trachea and esophagus)	1.8
Congenital pyloric defect	0.6
Other anomalies of the alimentary canal	2.6
Visual system:	
Congenital cataract	0.9
Congenital glaucoma	0.2
Other anomalies of the eye	2.9
Ear, face, and neck:	
Branchial cleft (vestigial, gill-like structure)	1.7
Anomaly of the ear with impaired hearing	0.6
Other anomalies of the ear (not all-inclusive)	4.4
Other anomalies of the face and neck	1.6
Musculoskeletal system:	
Hip dislocation without central nervous system defects	27.0
Clubfoot without central nervous system defects	24.5
Polydactyly (extra fingers or toes)	20.5
Skull and facial bone anomaly	12.8
Syndactyly (webbing between fingers or toes)	6.7
Arm anomaly N.E.C.[a]	5.0
Reduction deformity (absence of a portion or all of a body part, especially limbs)	3.5
Other congenital anomalies of the limbs (not all-inclusive)	92.8
Other musculoskeletal anomalies (not all-inclusive)	23.7
Other selected birth defects:	
Hemangioma of skin (birthmarks formed by bundles of blood vessels)	25.7
Anomalies of abdominal wall	7.0
Breast anomalies	2.6

[a] N.E.C.—Not elsewhere classified.

SOURCE: Birth Defects Monitoring Program.

Figure A-5.—Percentage of United States Live Births in the Birth Defects Monitoring Program (BDMP), by United States Census Region, 1980



SOURCE: U.S. Department of Health and Human Services, Centers for Disease Control, Congenital Malformations Surveillance, 1982.

and personal habits often contribute to the risk of being adversely affected by a given agent. Such confounding variables must be considered when attempting to isolate potential workplace hazards (134). In addition, the same agent may have different effects, depending on the extent and timing of embryo/fetal exposure. For example, structural abnormalities are most likely to be induced during the first 8 weeks of gestation (when many women do not yet know that they are pregnant), since this is when most differentiation and organogenesis takes place (see chapter 3). Later fetal stages may be equally sensitive to induction of certain other defects, such as carcinogenesis or behavioral, immunological, or endocrine abnormalities (111).

One study apportions the etiology of birth defects as follows (181):

- genetic transmission, 20 percent;
- chromosomal abnormalities, 5 percent;
- therapeutic radiation, 1 percent;
- infection, 2 to 3 percent;
- maternal metabolic imbalance (e.g., diabetes), 3 to 5 percent;
- drugs and environmental chemicals, 2 to 3 percent.

The remaining 63 to 67 percent of birth defects are designated as of unknown origin.

This section reviews the causal factors for major birth defects that have been identified to date, some only tentatively. These causal factors are categorized as environmental, pathological, heritable, iatrogenic, nutritional, or sociobehavioral.

Environmental Factors.—Various environmental pollutants, such as the ubiquitous polychlorinated biphenyls (PCBs), which are synthetic, chlorinated hydrocarbons used for electrical insulation (112,185), and possibly dioxins (119), are thought to produce birth defects. Although PCBs are transferred in greater amounts to the child postnatally through the mother's milk, prenatal transfer across the placenta may nonetheless have a significant impact on the embryo/fetus due to: 1) the continuous exposure, 2) the relatively small size of the fetus, 3) increased vulnerability at certain stages of intrauterine development, and 4) the fact that the fetus lacks the protective barriers found postnatally, such as the blood-brain barrier that bars certain drugs and potentially harmful substances from reaching the brain (65, see chapter 4).

Animal experiments suggest that exposure of the embryo/fetus in utero and neonatally to polycyclic aromatic hydrocarbons (major pollutants also found in cigarette smoke), radioactive substances, and gamma rays may destroy substantial numbers of female egg-cell precursors. Although there is presently no direct evidence, the human embryo/fetus may also be vulnerable to these agents, especially during the last trimester, leading to egg-cell-precursor depletion and eventual premature menopause (30,31). In the case of ionizing radiation, there is evidence that a dose of less than 10 rads to the implanted embryo does not significantly increase the incidence of congenital malformations, growth retardation, or fetal death (15a).

A recent report on more than 5,000 newborns found a dose-related correlation between high lead levels in the umbilical cord blood and increased risk of minor congenital anomalies such as hemangioma, hydrocele, and undescended testes (118).

Exposure of the embryo to the pesticide DDT (see chapter 4) before implantation in the uterus, and thus before protective placental barriers develop, may retard intrauterine growth, according to animal studies (36). In the mid-1960s, neurological disorders resembling cerebral palsy were discovered in children after maternal ingestion of mercury-contaminated fish in the now well-known case of Minamata, Japan (97; see chapter 2).

Maternal exposure to some pesticides used in farm work has been associated with diverse birth defects in isolated reports (46,59). There may also be a link between the consumption of high-nitrate groundwater by pregnant women and teratogenesis (33). Glycol ethers, a chemical species contained in a wide variety of products (see chapter 4)—including paints, stains, varnishes, and solvents—have been shown to be teratogenic in animals. High levels of air pollution have been associated with reduced birth weight (180).

Pathological Factors.—The first teratogenic infectious agent discovered was the rubella virus. This virus produces a variety of birth defects, including congenital cataracts, brain damage, and growth retardation (140) (see chapter 4). The only other infectious agents that have been unequivocally proven to be teratogenic are cytomegalovirus, the virus most commonly affecting human fetuses (although only 10 percent of those infected develop symptoms) (51) (see chapter 4), and a parasite, *Toxoplasma gondii*, transmitted through improperly cooked mutton or pork. These three maternal infections are estimated to cause an aggregate 2 percent of all congenital malformations (71). The agent of syphilitic infections, *Treponema pallidum*, is likely to be teratogenic if the pregnant woman is not treated for the infection. Other viruses and bacteria suspected of being teratogenic include: herpes simplex virus (116,142), varicella and herpes zoster (32), Venezuelan equine encephalitis (177), and influenza viruses (80). Other viruses that are not necessarily teratogenic but may cause fetal disease are mumps, polio, and hepatitis.

Heritable Factors.—The incidence of malformations among offspring of epileptic parents is two to three times higher than that in the general population. These malformations include cleft lip, cleft palate, and skeletal and cardiac abnormalities (151). Impaired mental function and increased perinatal mortality have also been associated with offspring of epileptic women. The problem is compounded by the fact that the anticonvulsant medications used to treat epilepsy may contribute to teratogenesis.

Maternal, insulin-dependent diabetes is associated with congenital malformations involving multiple organ systems, particularly the cardiovascular and musculoskeletal systems. There is an estimated two- to threefold increase in malformations among the offspring of diabetic women, constituting a rate of 6 percent or more. This effect is probably not seen in women whose diabetes is controllable by diet or oral hypoglycemic agents (113). Greater variability in maternal blood glucose has been strongly linked to a higher risk of neonatal complications, including stillbirth and metabolic blood imbalances (7). In recent years, the proportion of infants with congenital malformations born to diabetic mothers has increased, perhaps because of better management of the disease during pregnancy that allows the fetus to survive to a viable stage (122).

Maternal, sickle-cell anemia, afflicting 1 in 625 U.S. blacks (144), is associated with low birth weight and high perinatal mortality (45). Maternal phenylketonuria (a metabolic disorder caused by a deficiency of an enzyme needed to metabolize the amino acid phenylalanine, a normal protein component) is relatively rare, afflicting 1 in 12,000 people in the United States (144), but is associated with some serious defects including small head-size, growth retardation, heart defects, and childhood neurologic problems (66). Offspring of women with congenital heart disease may also exhibit higher perinatal mortality (34). The offspring of women with Down syndrome who did not inherit the syndrome have been reported to show higher frequencies of congenital malformations (135). A certain type of tumor causing secretion of hormones that induce hypertension in the mother can cause fetal death. Other rare tumors that cause maternal sex hormone imbalances may masculinize a female fetus (66).

Iatrogenic Factors.—Perhaps the most famous case of teratogenesis resulting from medical treatment involved thalidomide, a drug used widely in Europe during the early 1960s as a sedative (see chapters 2 and 3). The drug was relatively harmless to the mother but was belatedly found to produce severe limb deformities as well as anomalies of the heart, kidney, and gastrointestinal tract in the fetus when taken early in pregnancy (83,103).

Isoretinoin (Accutane®, Roche Laboratories, Nutley, NJ) an orally administered acne medicine licensed in 1982, was recently found to cause spontaneous abortion and a variety of birth defects when taken during the first trimester of pregnancy. The defects include small or absent outer ear and ear canal, central nervous system anomalies including hydrocephalus and microcephaly (small headsize), and congenital heart defects. Through mid-1984, there were 17 reported cases of birth defects and 20 reported instances of spontaneous abortion in women who were receiving isoretinoin. However, there is still no accurate information on the number of normal births among women using isoretinoin (95,154).

Some seemingly innocuous over-the-counter (OTC) drugs may also be hazardous because they are often regarded as nonpharmacological agents and are taken indiscriminately. One study found that 95 percent of pregnant women surveyed used OTC drugs during pregnancy. Some minerals and vitamins, the most frequently consumed OTC drugs (65 percent of women surveyed), can be fetotoxic in excess amounts. Excess maternal ingestion of Vitamin D may result in vascular disorders, mental retardation, and hypercalcemia. Chronic intake of analgesics, the second most frequent type of drugs consumed (61 percent), have been tentatively associated with low birth weight and stillbirth. Certain cough medications (ranking seventh at 11 percent) may also be hazardous: chronic maternal use of such codeine-containing compounds can cause symptoms similar to narcotics withdrawal in the newborn (61).

Other medicines now suspected, but not conclusively proven, to be teratogenic, include:

- **anticonvulsants,** used to treat epilepsy (38,186);
- **CNS stimulants,** such as certain amphetamines, the antidepressive imipramine, and possibly lithium (109,131);
- **sex hormones**: The synthetic estrogen diethylstilbestrol (DES), a drug used in the 1940s and 1950s to prevent miscarriage, is now known to be both teratogenic (73) and a transplacental carcinogen (60) (see chapter 2). Danazol, a pharmacologic used to treat endometriosis and fibrocystic breast disease, may also cause masculinization of the female fetus when taken during pregnancy, especially after the 8th week when the embryo becomes sensitive to danazol's male-hormone activity (127);
- **beta adrenergic drugs,** used to treat hypertension and cardiovascular disease (89);
- **anticoagulants,** such as warfarin or dicumarol, given orally during early pregnancy, are teratogenic (28,170) and increase the rate of spontaneous abortion and stillbirth (49);
- **alkylating agents** for the treatment of cancer have been associated with a variety of malformations (27,120), and intrauterine growth retardation and death (36);
- **antibiotics,** such as penicillins, tetracyclines, or chloramphenicol may be teratogenic or fetotoxic (16,17,130). Streptomycin may cause hearing loss and cerebral damage (172);
- **insulin and other antidiabetic drugs** (8);
- **antinauseants,** such as Bendectin® (Merrell-National Laboratories, Cayey, PR), and **antihistamines** (80); and
- **thyroid suppressants** (56).

Therapeutic X-rays at doses above 10 rads may harm the fetus; the risk at doses below this level becomes progressively minute (15b,15c). One hypothesis based on animal data suggests that chronic in-utero irradiation of the embryo/female fetus accumulating to above 20 rads may be associated with premature menopause in the offspring. Furthermore, there may be a period of particularly high sensitivity of the female fetal germ cells during the last trimester of gestation, during which comparable damage may be inflicted by a dose of about 7 rads (29). Exposure to radiation during the first 3 weeks of gestation is more likely to result in spontaneous abortion than other developmental effects (147).

Nutritional Factors.—Maternal malnutrition during pregnancy results in fetal growth retardation (182). The periods of famine during World War II were accompanied by increased rates of spontaneous abortion, stillbirth, neonatal death, and congenital malformation (5,136,137).

Although imbalances in various nutrients have been suspected of causing congenital malformations, only zinc deficiency has occurred often enough in humans to provide solid evidence of teratogenicity (18). Certain cytotoxic drugs used to treat cancer, as well as chronic alcohol consumption, can cause folate deficiency—a disorder related to CNS defects (140). Vitamin supplements taken soon after conception may help prevent CNS defects (141).

Sociobehavioral Factors.—Many recreational drugs (licit and illicit) are known to adversely affect the fetus. Alcohol is clearly teratogenic when consumed by the mother in large amounts (defined variably) and can result in "fetal alcohol syndrome," characterized by CNS dysfunction, mental retardation, growth deficiency, and facial deformities (72,151). Among neonates of alcoholic mothers, 83.3 percent had birth weights under the tenth percentile compared with 2.3 percent in a nonalcoholic sample (152). A prospective study of the relationship between birth weight and alcohol drinking during the first trimester of pregnancy in 31,604 pregnancies indicated that consuming at least 1 to 2 drinks daily was associated with a significantly increased risk of producing a growth-retarded infant. Conversely, consuming less than one drink daily had a minimal effect on intrauterine growth and birth weight. The authors note that "an occasional drink has only a trivial effect on intrauterine growth" (114).

Cigarette smoke and nicotine are also harmful, carrying an increased risk of: 1) prematurity, 2) low birth weight, due partly to fetal malnutrition resulting from depression of uterine circulation or maternal appetite, and 3) perinatal death (121,151). A pregnant woman who smokes two packs of cigarettes a day may reduce the oxygen supply to her fetus by 25 percent (4). Beginning in October 1985, new warning statements will be required (Public Law 98-474) on the packages and advertising of all cigarette brands sold in the United States (175). One of these statements calls specific attention to the hazards imposed by maternal smoking on the offspring:

> **SURGEON GENERAL'S WARNING:** Smoking by Pregnant Women May Result in Fetal Injury, Premature Birth, and Low Birth Weight.

Paternal smoking (35) and chronic alcohol consumption (84) are both associated with sperm abnormalities that may increase the risk of birth defects (66).

The regular use of marijuana has been linked to a shortened gestation period. A 1-week reduction in gestation length observed among heavy marijuana users is of questionable clinical significance in and of itself. However, the figure of 1 week is an average, and the reduction is more marked as the quantity consumed increases. The finding that marijuana usage can contribute to a shortened gestation length may take on clinical significance in certain individuals who consume large amounts of the drug or in individuals whose lifestyle habits include the use of other drugs that may also reduce the length of gestation (39).

Although caffeine is suspected of being a teratogen (13), heavy maternal coffee drinking (seven or more cups per day) is associated with increased risk of low birth weight offspring (62). Finally, narcotics can cause prematurity, retarded intrauterine growth, and neonatal addiction (151).

Advanced maternal age is considered a risk factor for a variety of birth defects, particularly for Down syndrome (87): the risk of bearing an affected child at age 45 is 1 in 32, more than 11 times greater than at age 35 (123). Since an estimated 25 percent of children with Down syndrome received their extra chromosome from their fathers (123), paternal risk factors should be further investigated. The incidence of Down syndrome may rise in the future due to the increasing popularity of delayed childbearing since the early 1970s. The first-birth rate in the 30 to 39 age group has risen markedly compared with younger age groups. The rate among women aged 30 to 34 doubled between 1972 and 1981 (159).

In contrast to infant mortality and low birth weight, birth defects are more common overall among white than black babies (165). Further, male babies and babies from plural deliveries (i.e., twins, triplets) are more likely to have birth defects. Finally, the incidence of birth defects is generally higher among babies born to women with less than a high school education (165).

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Appendix B

Sample Patient History Questionnaire

One of the primary and most useful tools in clinical practice is the patient history questionnaire. Information about an individual's medical, familial, occupational, and personal background can be critical to proper diagnosis and appropriate treatment of a medical condition. Moreover, written records may identify patterns of illness among individuals with a common lifestyle element.

A thorough, standardized patient information questionnaire could be particularly useful for recognizing patterns of work-related illness in the population. Epidemiological study of occupational disease is hampered by the fact that there is currently no validated or widely used questionnaire that gathers this information (8).

Consequently, the following section draws together segments of history questionnaires from various types of medical facilities (e.g., occupational medical centers, fertility clinics) in an effort to cover each of the categories that may be important for diagnosis and treatment. These include:

- identification (e.g., name, sex, age);
- occupational history (e.g., present and previous employment, exposures);
- lifestyle characteristics (e.g., use of nicotine and alcohol, exposures in home);
- familial health (e.g., medical conditions/diseases of relatives);
- medical history (e.g., injuries, medical conditions/ diseases, surgical procedures); and
- reproductive history (e.g., reproductive difficulties or disorders, past reproductive outcomes)

Since this questionnaire is a composite of questionnaires from a broad range of clinical and research facilities, it is not validated for use. It was developed solely to inform the reader of the number of factors that are pertinent to a thorough understanding of a patient's medical and personal background. Specific investigators would likely select a subset of variables that relate to the reproductive endpoints being studied.

Appendix B References

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PATIENT HISTORY QUESTIONNAIRE

I. IDENTIFICATION

Name:__________________

Social Security:____________

Address:________________

Sex: M F
Birthday:________________

Telephone: home____________
work____________

Height:__________________
Weight:__________________

II. OCCUPATIONAL HISTORY

A. Present Employment

WHICH OF THE FOLLOWING BEST DESCRIBES YOUR CURRENT JOB STATUS? (PLEASE CHOOSE ONLY ONE)

A. Employed full time __________ If so, since what year? __________
B. Employed part time __________ If so, since what year? __________
C. Multiple jobs __________ If so, since what year? __________
D. Retired __________ If so, since what year? __________
E. Disabled __________ If so, since what year? __________
F. Unemployed __________ If so, since what year? __________
G. Laid off __________ If so, since what year? __________
H. Student __________ If so, since what year? __________
I. Homemaker __________ If so, since what year? __________

IF YOU ARE PRESENTLY EMPLOYED, WHAT IS YOUR JOB? HOW LONG HAVE YOU BEEN SO EMPLOYED? ______________________________

1. WHAT HAS BEEN YOUR USUAL OCCUPATION OR JOB — THE ONE YOU HAVE WORKED AT THE LONGEST?

A. Job/Occupation (e.g.,carpenter, homemaker) ______________________

____

B. Number of years in this occupation ______________________

C. What kind of business or industry is this? (e.g.,hospital, ship building)

____

note which of the following types of equipment you use, and about how much of the time that you actually use it of the time that you think you should (for example, you may find a mask respirator uncomfortable and wear it only about half the time that you think you should be wearing it)

Mark if used at all.

If used, about what part of the time is it used that you think it should be used:

	Less than 1/4	About half	About 3/4	All the time
☐ Mask respirator				
☐ Air supply respirator				
☐ Gloves				
☐ Coveralls or aprons				
☐ Safety glasses				
☐ Hearing protection				
☐ Other (identify)				

PLEASE CHECK OFF THE FOLLOWING REGARDING ASPECTS OF THIS JOB.

	Yes	No
A. Use separate workclothes	☐	☐
B. Use separate shoes	☐	☐
C. Has a lunchroom removed from work exposures	☐	☐

HOW MUCH HARD PHYSICAL WORK IS REQUIRED ON YOUR JOB — LIKE PUSHING OR CARRYING HEAVY OBJECTS, HANDLING HEAVY TOOLS OR EQUIPMENT, OR DIGGING?

☐ A great deal ☐ Some ☐ Hardly any ☐ None at all

HOW WOULD YOU DESCRIBE THE DEGREE OF EMOTIONAL STRESS ASSOCIATED WITH THIS JOB?

☐ A Great Deal ☐ Some ☐ Hardly any ☐ Don't know

IN TERMS OF THE AMOUNT OF STRESS ON THIS JOB, HOW WOULD YOU COMPARE IT WITH OTHER JOBS YOU HAVE HAD?

☐ Much less ☐ About the same ☐ A bit more ☐ A great deal more

WERE YOU EVER GIVEN JOB SAFETY/HEALTH TRAINING FOR THIS JOB?

☐ Yes ☐ No

If yes, by whom?

☐ Management ☐ Union ☐ Other (specify) ______________________

IN THIS JOB, HAVE YOU HAD PREEMPLOYMENT OR PERIODIC EXAMS FOR ANY HAZARD-RELATED HEALTH PROBLEMS?

☐ Yes ☐ No

If yes, have you ever been told that these exams were abnormal and if so describe.

____

____

____

B. Employment History

FILL IN THE TABLE BELOW LISTING ALL JOBS AT WHICH YOU HAVE WORKED, INCLUDING SHORT-TERM, SEASONAL, AND PART-TIME EMPLOYMENT. START WITH YOUR PRESENT JOB AND GO BACK TO THE FIRST. USE ADDITIONAL PAPER IF NECESSARY.

Workplace (Employer's name and address or city)	Dates worked		Did you work full time?	Type of Industry (Describe)	Describe your job duties	Known health hazards in workplace (dusts, solvents, etc.)	Protective equipment used?	Were you ever c work for a heal problem or injur
	From	To						

C. Exposure History

HAVE YOU EVER WORKED AT A JOB OR HOBBY IN WHICH YOU CAME INTO DIRECT CONTACT WITH ANY OF THE FOLLOWING SUBSTANCES BY BREATHING, TOUCHING, OR DIRECT EXPOSURE? IF SO, PLEASE CHECK THE BOX BESIDE THE SUBSTANCE.

The questions below are an important part of our evaluation of your problem. Below is a list of agents or exposures that you may have encountered in your work or outside work.

The first set of boxes — marked A — refers to your current or most recent job (job #). For any agent or exposure that you have worked with in this job, mark YES and whether you think the exposure was of low, medium, or high amount.

Do the same for the next set of boxes — marked B — which refer to any previous job (any job aside from job #1). And then do the same for the last set of boxes — marked C — which refer to any activities outside paid work, such as housekeeping, student activities, and hobbies.

LIST OF EXPOSURES	YES	IF YES CHECK ONE			YES	IF YES CHECK ONE			YES	IF YES CHECK ONE		
		Low	Med	High		Low	Med	High		Low	Med	High
Example Asbestos	✓	✓			✓			✓				
1. FUMES AND DUSTS												
Asbestos												
Plastic Fumes												
Welding Fumes												
Fumes (other)												
Glass (e.g. Fiberglass)												
Silica (e.g. Sand)												
Plaster												
Wood (Specify Type(s) If Known:____________)												
Other (Specify If Known:____________)												

	A. Current or Most Recent Job (Paid Work)				**B. Any Previous Job**				**C. Any Activity Outside Paid Work**			
	YES	IF YES CHECK ONE			YES	IF YES CHECK ONE			YES	IF YES CHECK ONE		
		Low	Med	High		Low	Med	High		Low	Med	High
2. ELEMENTS AND METALS												
Aluminum												
Arsenic												
Cadmium												
Chromium												
Copper												
Lead												
Mercury												
Nickel												
Zinc												
Other (Specify If Known: ______)												
3. SOLVENTS												
Alcohols (e.g. Methyl, Wood)												
Benzine (Gas), Petroleum Ether												
Benzene, Toluene, Xylene												
Carbon Tetrachloride												
Paint, Varnish, Degreasers												
Tri-, Tetrachloroethylene												
Other (Specify If Known: ______)												
4. OTHER CHEMICALS												
Acids												
Alkali (Caustics)												
Ammonia												
Detergent and Soaps												
Dyes												

	A. Current or Most Recent Job (Paid Work)				B. Any Previous Job				C. Any Activity Outside Paid Work			
	YES	IF YES CHECK ONE			YES	IF YES CHECK ONE			YES	IF YES CHECK ONE		
		Low	Med	High		Low	Med	High		Low	Med	High
Formaldehyde												
Pesticides												
Plastic Resins												
Other (Specify If Known: ________)												
5. MISCELLANEOUS												
Heavy Lifting												
Improper Lighting												
Excess Heat or Cold												
Emotional Stress												
Plant Products												
Ionizing Radiation (e.g., X-ray, Radioisotopes)												
Nonionizing Radiation (e.g., Microwave, UV)												
Noise												
Sitting or Standing in Same Position												
Vibration												
Other (Specify If Known: ________)												

D. Occupational Illness

1. Please describe any health problems or injuries you have experienced connected with your present or past jobs.

HOW MANY PEOPLE WORK WITH YOU IN YOUR IMMEDIATE AREA?

☐ 0-5 ☐ 6-10 ☐ 11-25 ☐ 25-100 ☐ Greater than 100

3. Have any of your co-workers also experienced health problems or injuries connected with the same jobs? If yes, please describe.

DID YOU EVER CHANGE JOBS BECAUSE YOU WERE CONCERNED ABOUT OCCUPATIONAL HAZARDS OR DANGERS TO YOUR HEALTH?

☐ Yes ☐ No

HAVE YOU EVER BEEN DIAGNOSED AS HAVING A WORK-RELATED ILLNESS OR DISEASE?

☐ Yes ☐ No

If yes, please describe ____________________

____

____

If yes, who made the diagnosis? ☐ Self ☐ Own M.D.

☐ Company M.D. or nurse ☐ Other (specify) ____________________

HAVE YOU EVER HAD AN OCCUPATIONAL INJURY OR ILLNESS WHICH RESULTED IN A PERMANENT CHANGE OF JOB OR A TEMINATION OF A JOB?

☐ Yes ☐ No

HAVE YOU EVER HAD AN OCCUPATIONAL INJURY/ILLNESS WHICH RESULTED IN A LOST WORKDAY (one in which you could not work or were assigned to a different job)?

☐ Yes ☐ No

If yes, please describe ____________________

____

If yes, about how many workdays have you had in the last five years? ______

HAVE YOU EVER HAD AN OCCUPATIONAL INJURY/ILLNESS WHICH DID NOT RESULT IN A LOST WORKDAY BUT REQUIRED MEDICAL TREATMENT?

☐ Yes ☐ No

III. LIFESTYLE CHARACTERISTICS

1. DO YOU LIVE NEXT DOOR TO OR VERY NEAR AN INDUSTRIAL PLANT? YES NO
If so, please describe:

2. HAVE YOU EVER CHANGED YOUR RESIDENCE OR HOME BECAUSE OF A HEALTH PROBLEM? YES NO
If so, please describe:

3. DOES YOUR SPOUSE OR ANY OTHER HOUSEHOLD MEMBER HAVE CONTACT WITH DUSTS OR CHEMICALS AT WORK OR DURING LEISURE? YES NO
If so please describe:

DO YOU HAVE ANY HOBBIES?

☐**Yes** ☐**No**

If yes, list and estimate the number of hours per month you spend on each:

Hobby	**Hours**

5. DO YOU USE PESTICIDES AROUND YOUR HOME OR GARDEN? YES NO
If so, please describe:

6. WHICH OF THE FOLLOWING DO YOU HAVE IN YOUR HOME?
_Air Conditioner _Air Purifier _Humidifier _Electric Stove
_Fireplace _Central Heating

7. DO YOU OR HAVE YOU EVER SMOKED CIGARETTES, CIGARS, OR PIPES? YES NO
If so, how many per day:

8. ALCOHOL—APPROXIMATE NUMBER OF SERVINGS PER WEEK _____
Type of Beverage: ________________

9. DO YOU OR HAVE YOU EVER USED MARIJUANA? YES NO
If so, in what amounts?____________________

10. DO YOU OR HAVE YOU EVER USED

Cocaine	YES NO
Hallucinogens (e.g., LSD)	YES NO
Downers (e.g., sleeping pills)	YES NO
Uppers (e.g., pep pills)	YES NO
Heroin or other hard drugs	YES NO

11. ARE YOU OR HAVE YOU EVER BEEN DRUG AND/OR ALCOHOL DEPENDENT? YES NO
If so, on which drugs are/were you dependent? For how long?

IV. FAMILY HISTORY

1. HAS ANY BLOOD RELATIVE HAD ANY OF THE FOLLOWING?
Include Father, Mother, Brothers, Sisters, Grandparents, Aunts, Uncles, 1st Cousins

YES	NO	
		Anemia or low blood
		Arthritis
		Arteriosclerosis
		Asthma
		Autoimmune Disease (e.g Lupus, Ulcerative Colitis, Scleroderma
		Cancer
		Cystic fibrosis
		Easy bleeding
		Endocrine disorder (e.g. Goiter, Hyperthyroidism
		Glaucoma, Blindness, Cataracts
		High blood pressure (Hypertension)
		Hay fever, pollen allergies, eczema
		Heart disease
		Hodgkins
		Kidney disorders
		Leukemia
		Muscular distrophy
		Neurologic disorders (e.g. Parkinsons, Epilepsy, Multiple sclerosis)
		Sickle cell anemia
		Stroke
		Sugar diabetes
		Tay Sachs
		Tuberculosis (TB)

V. MEDICAL HISTORY

1. DO YOU CONSIDER YOUR GENERAL HEALTH:
   Poor__ Fair__ Good__ Excellent__

2. DO YOU CONSIDER YOUR GENERAL DISPOSITION:
   Calm__ Nervous__ Irritable__
   Depressed__ Happy__ Other__

3. HOW WOULD YOU CHARACTERIZE THE AMOUNT OF STRESS IN YOUR LIFE:
   Not Stressful__ Average__ Extraordinary__

4. DO YOU HAVE ANY ALLERGIES OR ALLERGIC CONDITIONS? YES NO
   If so, please describe:

5. LIST ALL OF THE MEDICATIONS YOU ARE TAKING INCLUDING THOSE THAT DO NOT REQUIRE A PRESCRIPTION. (e.g. Vitamins, Minerals, Aspirin)

Name of Medicine	Amount

6. ARE YOU ALLERGIC OR HAVE YOU HAD A "BAD REACTION" TO ANY MEDICATIONS? _Yes _No _Don't know
   If yes, list the medications and
   reactions ______________________________

7. HAVE YOU INCURRED ANY INJURIES (e.g. broken bones, burns, head injuries)?
   State any residual deformity or impairment.

8. DO YOU HAVE OR HAVE YOU HAD ANY OF THE FOLLOWING

YES	NO	
		Anemia
		Asthma
		Bladder infections
		Bronchitis
		Cancer
		Chicken pox
		Duodenal Ulcer
		Dysentery
		Endocrine disorder (goiter, hyperthyroidism)
		Epilepsy
		Hay fever or grass and tree allergies
		Heart murmer
		Heart disease
		High blood pressure
		Kidney disease
		Liver disease, jaundice, hepatitis
		Long term bowel trouble
		Malaria

Measles
Mental troubles
Mumps
Pneumonia
Rheumatic fever
Serious injury or accident
Sinus trouble
Stomach ulcer
Sugar diabetes
Tuberculosis
Typhoid
Uncontrolled bleeding
Venereal Disease

9. LIST ALL HOSPITALIZATIONS YOU HAVE HAD:

Type of illness/operation	Year
____	______
____	______
____	______
____	______

[illegible]. SYMPTOMS: PLEASE MARK (X) IN THE AVAILABLE BLANKS IF ANY OF THE FOLLOWING APPLY TO YOU *NOW* OR IN THE *PAST 3 MONTHS*. FOR ANY SYMPTOM THAT YOU MARK, CHECK WHETHER THIS SYMPTOM IS *BETTER*, *WORSE*, OR *NO DIFFERENT* WHEN YOU ARE AT WORK.

	MARK IF PRESENT NOW OR IN PAST 3 MONTHS	BETTER	WORST	NO DIFFERENCE	DON'T KNOW
HEAD, EYES, EARS, NOSE, THROAT	↓				
Dizziness					
Severe headaches					
Double vision					
Poor eyesight					
Ear or hearing trouble					
Frequent nose trouble					
Persistent hoarseness					
Teeth trouble					
Sore mouth					
Eye trouble					
Funny taste in mouth					
Ringing in ears					
Runny nose					
LUNGS	↓				
Daily cough					
Daily coughing of phlegm (mucous)					
Coughing blood					
Persistent wheezing					
Shortness of breath					
Chest pain when breathing					
HEART	↓				
Chest pain when walking					
Heart palpitation (fluttering, skipping, going fast)					
Leg vein trouble					
Leg pain when walking					
Ankle swelling					

STOMACH, INTESTINAL

	MARK IF PRESENT NOW OR IN PAST 3 MONTHS ↓	BETTER	WORST	NO DIFFERENCE	DON'T KNOW
Trouble swallowing					
Frequent or severe nausea					
Frequent or severe heartburn					
Frequent indigestion					
Frequent or severe stomach pain					
Frequent or severe vomiting					
Vomiting blood					
Yellow jaundice					
Bowel habit change					
Prolonged or frequent diarrhea (bowel movements)					
Constipation					
Blood in bowel movements					
Black bowel movements					
Hemorrhoids (piles)					

URINARY

	↓				
Frequent urination					
Painful urination					
Bloody urine					
Trouble starting urine					
Urinate more than 2 times a night					
Trouble holding urine					

BONES, JOINTS, MUSCLES

	↓				
Joint pains and swelling					
Severe lack of strength					

GENERAL	MARK IF PRESENT NOW OR IN PAST 3 MONTHS ↓	BETTER	WORST	NO DIFFERENCE	DON'T KNOW
Unexplained weight loss or gain					
Unexplained fever					
Night sweats					
Can't stand hot weather					
Can't stand cold weather					
Persistent skin rash or itching					
Increased sweating					

NERVOUS SYSTEM	↓				
Lack of energy					
Frequent loss of balance					
Fainting spells (black outs)					
Convulsions (seizures, fits, epilepsy)					
Tremor (shaking, trembling)					
Paralysis					
Numbness (body parts "go to sleep")					
Nervousness					
Excessive worry					
Trouble sleeping					
Memory trouble					
Trouble concentrating					
Depression (feeling blue)					
Crying spells					
Feelings of worthlessness					
Trouble getting along with people					
Pins and needles, funny sensations					

13. SYMPTOMS (cont.)

MALES	**MARK IF PRESENT NOW OR IN PAST 3 MONTHS** ↓	BETTER	WORST	NO DIFFERENCE	DON'T KNOW
Discharge from penis					
Testicles (balls) trouble					
Sexual trouble					

FEMALES					
Breast lumps or discharge					
Unusual bleeding from vagina (birth canal)					
Unusual discharge from vagina (birth canal)					
Sexual trouble					

14. HAVE YOU OR YOUR SPOUSE (OR PARTNER) HAD ANY DIFFICULTY IN BECOMING PREGNANT? ☐Yes ☐No

15. DO YOU HAVE ANY OTHER HEALTH PROBLEM THESE QUESTIONS HAVE MISSED? ☐Yes ☐No

If yes, please list ______________________________

____

16. IN YOUR OPINION, WHAT ARE YOUR MOST IMPORTANT HEALTH PROBLEMS? LIST AS MANY AS YOU CAN.

1. ______________________________
2. ______________________________
3. ______________________________

VI. REPRODUCTIVE HEALTH[*]

A. MALE

1. HAVE YOU EVER HAD ANY INJURY OR OPERATION TO THE PENIS OR TESTICLES?
Circumcision YES NO
Other operations on penis YES NO
Varicocele operation (varicose veins near testicles) YES NO
Vasectomy YES NO
Biopsy of the testicle YES NO
Other operations or injuries to the testicles YES NO

2. HAVE YOU EVER HAD AN INFECTION OF THE
Bladder YES NO
Urethra YES NO
Epididymis YES NO
Kidney YES NO
If so, please give details:

3. HAS THERE BEEN ANY RECENT CHANGE IN THE SIZE OF YOUR TESTICLES? YES NO
If so, please give details:

4. HAVE YOU EVER HAD A HERNIA OPERATION (Even as a baby)? YES NO
If so, please give details:

5. ARE YOU IN THE HABIT OF TAKING VERY HOT BATHS? YES NO

6. ARE YOU IN THE HABIT OF TAKING SAUNAS? YES NO

7. WHAT SORT OF UNDERWEAR DO YOU NORMALLY WEAR?
____Boxer trunks
____Jockey shorts
____Other
____

8. HAVE YOU EVER BEEN TOLD BY A DOCTOR THAT YOU HAD A PROSTATE PROBLEM? YES NO

9. HAVE YOU EVER GONE THROUGH A PERIOD OF SEVERAL MONTHS WHEN YOU HAD TROUBLE GETTING OR KEEPING AN ERECTION? YES NO
If so, please give details:

10. DO YOU GET SATISFACTORY EJACULATION OF SPERM DURING INTERCOURSE? YES NO

11. HAVE YOU EVER GONE THROUGH A PERIOD OF SEVERAL MONTHS WHEN YOU HAD LITTLE INTEREST IN SEX? YES NO
If so, please give details:

---

[*] This section is designed specifically for the fertility patient. Certain questions are, therefore, unnecessary for a standard patient history form.

12. DO YOUR HAVE ANY PROBLEMS URINATING? YES NO

13. HAVE YOU EVER BEEN EXAMINED BY A UROLOGIST? YES NO
If so, when?________ For what reason?______________
Were any problems identified?_________________________

14. HAVE YOU EVER ATTENDED AN INFERTILITY CLINIC OR HAD PREVIOUS TREATMENT FOR INFERTILITY? YES NO
If so, please give name of the doctor and the facility:
____

15. IS THERE ANY HISTORY OF FERTILITY PROBLEMS IN YOUR FAMILY?
(difficulty conceiving, miscarriage, still birth, deformed offspring)
Parents?____
Brothers?____
Uncles?____

16. HAS YOUR SEMEN BEEN EVALUATED BEFORE? YES NO
How many times?___________
When most recently?_________
What were the results?______________________________
Have other tests (e.g. antibody tests, mucus penetration) been done with your semen? YES NO
If so, when?_______________
What were the results?______________________________

17. HAVE ANY ENDOCRINE (HORMONE) STUDIES BEEN DONE WITH YOUR BLOOD? YES NO
If so, when?_______________
What were the results?______________________________

18. HAVE YOU AND YOUR PRESENT OR ANY PREVIOUS MATE HAD DIFFICULTY CONCEIVING? (unprotected intercourse for a year or more with no pregnancy) YES NO

19. HAVE YOU FATHERED A PREGNANCY THAT ENDED IN ANY OF THE FOLLOWING?
If so, please specify whether it was with your present or a previous mate:

__Miscarriage
__Stillbirth
___Baby born more than 2 weeks early
___Baby with a birth defect:
  __Cleft palate
  __Harelip
  __Limb deformity
  __Disease or deformity of the heart, lungs, kidney, genitals, urinary tract, gastrointestinal tract,
__Twins/Multiple offspring
__Low birth weight ($5^1/_2$ pounds or less)

nervous system

___Malformations of the skull, spine

___Musculoskeletal disorders (e.g. muscular distrophy

20. HAVE YOU FATHERED ANY CHILDREN WHO HAVE ANY OF THE FOLLOWING CONDITIONS?

Please specify whether these children were born to you and your present or a previous mate:

___Allergy

___Asthma

___Epilepsy

___Downs syndrome

___Cystic fibrosis

___Hemophilia

___Mental retardation or learning problem

___Leukemia

___Tumor or Cancer

___Tay-sachs

___Cerebral palsy

___ Other (specify)

B. FEMALE

MENSTRUAL HISTORY:

1. HOW OLD WERE YOU WHEN YOU BEGAN TO MENSTRUATE?________

2. ARE YOUR PERIODS REGULAR? YES NO

3. WHAT IS THE AVERAGE LENGTH OF YOUR CYCLE?_____________

4. GIVE THE DATE OF THE 1ST DAY OF YOUR LAST PERIOD:_________

5. GIVE THE DATE OF THE 1ST DAY OF THE PERIOD BEFORE LAST:_____

6. FOR HOW MANY DAYS DO YOU LOSE BLOOD?_________________

7 IF YOU EXPERIENCE ANY OF THESE SYMPTOMS, NOTE HOW MANY DAYS BEFORE ONSET OF BLEEDING THE SYMPTOM BEGINS:

Premenstrual:
Abdominal Bloating___
Swelling of face, hands or feet_____
Breast Tenderness_____
Weight Gain________
Bowel Changes_______
Urinary Tract Symptoms_____
Headache______________
Irritability____________
Other_________________

During Period:
Cramps___________
Nausea___________
Diarrhea__________
Chills____________
Headaches__________
Fainting, Dizziness____
Hot Flashes_____________
Fever________________
Sweats________________
Constipation____________
Rectal Pain_____________
Other__________________

8. DO YOU HAVE ANY BLEEDING OR BLOODY DISCHARGE:

Between Periods	YES NO
After Intercourse	YES NO
After Douching	YES NO

CONTRACEPTION:

1. DO YOU USE OR HAVE YOU USED ANY OF THE FOLLOWING TYPES OF CONTRACEPTION?
Oral contraceptive pill___
Diaphragm___________
Condom_____________
Spermacidal foam or gel___
Permanent sterilization___
Tubal ligation________
Coitus interruptus_______
IUD_________________

2. WHAT FORM OF CONTRACEPTION, IF ANY, ARE YOU CURRENTLY USING?

GYNECOLOGIC HISTORY:

1. DO YOU HAVE ANY PAIN OR DISCOMFORT ASSOCIATED WITH INTERCOURSE? YES NO

2. DO YOU HAVE ANY PROBLEMS OR DIFFICULTY RELATED TO SEXUAL ACTIVITY? YES NO

3. HAVE YOU HAD GENITAL HERPES? YES NO

4. HAVE YOU HAD VENEREAL DISEASE? YES NO

5. HAVE YOU EVER HAD AN ABNORMAL PAP SMEAR? YES NO

6. HAVE YOU HAD OR DO YOU HAVE RECURRENT VAGINAL INFECTION? YES NO

7. HAVE YOU HAD OR DO YOU HAVE PROBLEMS WITH VAGINAL DISCHARGE? YES NO

8. DID YOUR MOTHER TAKE *DES* WHILE PREGNANT WITH YOU? YES NO

9. HAVE YOU HAD ANY TYPE OF PELVIC INFECTION, DISEASE, ABNORMALITY OR SURGERY OF THE
   Vulva___ Vagina___ Cervix___
   Uterus___ Tubes____ Ovaries___
   Urinary Tract__ Anus_____ Rectum_____

10. HAVE YOUR EVER HAD ENDOMETRIOSIS?
    If so, when?______ How was is treated?____________________________

11. ARE YOUR FALLOPIAN TUBES OPEN? YES NO

12. HAS EITHER TUBE BEEN REMOVED? YES NO

13. HAVE YOU EVER HAD A HYSTEROSALPINGOGRAM (tubal dye study)YES NO
    If so, when?_______ What were the results?________________________

14. HAVE YOU EVER HAD A LAPAROSCOPY? YES NO
    If so, when?_______ What were the results?_________________________

13. HAVE YOU EVER HAD A FERTILITY INVESTIGATION? YES NO
    If so, what was the diagnosis?
    _____Anatomical defect
    _____Hormonal/Glandular disorder
    _____Other
    _____No abnormality found

14. HAVE YOU EVER HAD SURGERY FOR INFERTILITY? YES NO
    If so, give details:

REPRODUCTIVE HISTORY:

1. ARE YOU MARRIED? YES NO

2. HAVE YOU BEEN MARRIED PREVIOUSLY? YES NO
   If so, how many times?__________

3. HOW LONG HAVE YOU BEEN TRYING FOR A PREGNANCY WITH YOUR PRESENT MATE? YES NO

4. HOW MANY TIMES PER WEEK DO YOU HAVE SEXUAL INTERCOURSE WITH YOUR PRESENT MATE? ______

5. DO YOU TRY TO HAVE INTERCOURSE DURING THE FERTILE TIME OF THE MONTH? YES NO
If so, how do you decide that the best time is?

6. DO YOU HAVE ANY PHYSICAL DIFFICULTIES WITH SEX THAT WOULD PREVENT A CONCEPTION (e.g. pain during intercourse sufficient to prevent penetration)? YES NO

7. DO YOU USE LUBRICANTS DURING SEXUAL INTERCOURSE? YES NO

8. HAVE YOU EVER GONE THROUGH A PERIOD OF SEVERAL MONTHS WHEN YOU HAD LITTLE INTEREST IN SEX? YES NO
If so, give details:

9. HAVE YOU AND YOUR PRESENT MATE EVER HAD A POST COITAL TEST (examination of the cervix for sperm after intercourse)? YES NO
If so, was any incompatibility noted?__________________

10. HAVE THERE BEEN ANY PREGNANCIES DURING THIS MARRIAGE?YES NO
If so, when did they occur?

11. HAVE THERE BEEN ANY MISCARRIAGES, ECTOPIC PREGNANCIES OR STILLBIRTHS DURING THIS MARRIAGE? YES NO
If so, when did they occur?

12. HAVE YOU EVER HAD A PREGNANCY THAT RESULTED IN ANY OF THE FOLLOWING?
If so, please specify whether it was with your present or a previous mate:
__Low birth weight baby (less than 5 $^{1}/_{2}$ pounds)
__Baby born more than 2 week early?
__Twins, triplets, etc.
__Baby with a birth defect:
__Cleft palate
__Harelip
__Limb deformity
__Disease or deformity of the heart, lungs, kidney, genitals, urinary tract, gastro-intestinal tract, nervous system
__Malformations of the skull, spine
__Musculoskeletal disorders (e.g. muscular distrophy

distrophy

13. HAVE YOU GIVEN BIRTH TO CHILDREN WHO HAVE ANY OF THE FOLLOWING CONDITIONS?
Please specify whether these children were born to you with your present or a previous mate.

___Allergy
___Asthma
___Epilepsy
___Downs syndrome
___Cystic fibrosis
___Hemophilia
__Mental retardation or learning problem
___Leukemia
___Tumor or Cancer
___Tay-sachs
___Cerebral palsy
___Other (specify)

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Appendix C

Technical Notes: OSHA

Technical Note #C.1: Medical Surveillance Programs: Questions and Answers

In implementing medical surveillance programs a number of questions have arisen, including the following:

(1) What employees are covered by the medical surveillance provisions of OSHA standards?

As mentioned in chapter 7, some OSHA health standards require medical surveillance only for employees exposed at or above the action level specified in the standards. Other standards require medical surveillance for all employees exposed to any levels of the substance. Even these more stringent requirements have been upheld. In *GAF Corp. v. OSHRC*,[1] the D.C. Circuit affirmed the Commission's holding that the employer was required to provide medical examinations for all employees exposed to asbestos, including employees whose exposures were below the PEL. In *Duquesne Light Co.*,[2] however, the Commission recently held that the asbestos standard did not require medical examinations of employees who were not regularly exposed, even though their sporadic exposures sometimes exceeded the standard. The coke oven, arsenic, and ethylene oxide standards require medical surveillance for employees exposed at least 30 days per year.

In formulating the ethylene oxide standard, OSHA rejected the recommendations of AFSCME and the AFL-CIO that medical surveillance should be provided to all formerly exposed employees as well as those presently exposed. According to OSHA, this recommendation was rejected because the present state of knowledge about ethylene oxide's long-term effects on humans is inadequate and only employees at a late stage of developing leukemia could be identified. The coke oven emissions standard, however, does require continued surveillance of previously exposed employees who have been reassigned by the same or a successor employer.[3]

(2) Are medical examinations mandatory?

Section 6(b)(7) of the Act provides that medical examinations shall "be made available" to exposed employees. OSHA has interpreted this language to mean that the employer must offer the examination; the employee may refuse to take the examination.[4] The coke oven emissions standard contains a provision requiring employers to inform employees of the possible health consequences of refusing to take the examination and requiring a signed statement by the employee that the consequences have been explained and understood.[5]

The detailed medical removal policy (MRP) and rate retention (RR) provisions of the lead standard were promulgated, in part, as an alternative to mandatory worker participation in the medical surveillance program. The preamble to the lead standard indicates that OSHA rejected the idea of making examinations mandatory because employees concerned about job security might be tempted to use chelating drugs as well as to conceal subjective symptoms of lead disease.[6] By contrast, with MRP and RR, workers would be encouraged to participate, but those who choose not to—because of privacy, religious, or other reasons—would not be required to participate.

The only time OSHA attempted to make medical surveillance mandatory was in the commercial diving standard, which was issued in 1977 and struck down by the Fifth Circuit in 1979.[7] OSHA reasoned that the safety of other dive team members can depend on the health of an individual diver.[8] The multiple-physician review procedure, discussed in detail below, also was included in the diving standard to ensure that divers would not be denied their employment on the basis of a single medical examination.

The preceding discussion of the "optional" nature of OSHA-required medical examinations does not mean that adverse consequences will not attach when an employee refuses to undergo examination. Simply because OSHA does not require participation does not mean that it protects a refusal to participate. Unless covered by the terms of a collective bargaining agreement, an employer may make cooperation with medical examinations a valid condition of employment.[9] Thus, as a practical matter, most "optional" OSHA medical examination provisions may be, in fact, mandatory for employees if the employer chooses to make them so.

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[1] 561 F.2d 913 (D.C. Cir. 1977).

[2] 11 O.S.H. Cas. (BNA) 2033 (1984).

[3] 29 C.F.R. § 190.1029(j)(3)(iii) (1984).

[4] B. Mintz, OSHA: History, Law, and Policy 134 (1984).

[5] 29 C.F.R. § 1910.1029 (j)(1)(iii) (1984).

[6] 43 Fed. Reg. 52,952, 52,973-74 (1978).

[7] Taylor Diving & Salvage Co. v. U.S. Dept. of Labor, 599 F.2d 622 (5th Cir. 1979).

[8] 42 Fed. Reg. 37,656-57 (1977).

(3) What procedures are required?

OSHA's health standards prescribe the specific medical procedures required during OSHA-mandated medical examinations. The argument has been made that broader latitude should be given the examining physician by adopting more performance-oriented standards. This would allow physicians to change their practices quickly to comport with the latest medical developments. In rejecting this argument in the ethylene oxide standard, OSHA's preamble noted that mandatory requirements help smaller employers with less established medical departments to determine the appropriate examination protocols.

Even without a separate health standard specifying the particulars of a medical surveillance program, OSHRC may impose an appropriate medical surveillance program as an alternative measure during the extended period of time request in a petition for modification of abatement (PMA). In *ITT* v. *Grinnell*,[10] the employer was cited for having excessive levels of silica dust. The employer filed a PMA to extend the abatement date, which the Commission granted conditioned on the employer's use of additional medical surveillance, including chest X-rays and pulmonary function tests.

Although OSHA prescribes the use of specific medical procedures, it should be emphasized that OSHA does not prohibit the use of any procedures. The only exception to this principle is the ban on the use of prophylactic chelation in the lead standard.[11]

(4) How are test results interpreted?

An accurate medical assessment often depends on a thorough history, clinical evaluation, and laboratory procedures. Although OSHA health standards promulgated after the asbestos standard (OSHA's first health standard promulgated by rulemaking) have contained appendixes with medical surveillance guidelines,[12] only the lead and cotton dust standards provide detailed guidance for physicians. The medical surveillance guidelines published with the proposed ethylene oxide standard recommended the use of cytogenetic monitoring of workers to detect chromosomal aberrations.[13] This recommendation was not included when the final version of the standard was promulgated.[14]

(5) Who selects the physician?

The Act does not specifically indicate whether the employer or employee has the right to select the physician who performs medical examinations. In promulgating the asbestos standard, OSHA determined that the employer should have the option of choosing the physician and should have access to the results of the examination.[15] The D.C. Circuit upheld OSHA's position[16] and this policy has been followed in subsequent health standards.

A notable exception concerns the "multiple physician review" procedure, first used in the commercial diving standard. The standard required medical examination of employees who were to be exposed to hyperbaric conditions. If the employee was found to be unfit by the examining physician selected by the employer, the employee could seek a second opinion. If the first two physicians disagreed, a third physician was to be selected by the first two physicians and that physician's determination would be dispositive. All costs were to be borne by the employer.

In *Taylor Diving & Salvage Co.* v. *U.S. Department of Labor*,[17] the Fifth Circuit struck down this provision. The court, citing its decision in *American Petroleum Institute* v. *OSHA*,[18] held that the standard was not "reasonably necessary or appropriate to provide safe or healthful workplaces." The court concluded that the standard imposed a mandatory job security provision controlled by the third physician. "[T]he employer has no control over the third doctor's fitness standards, so that the employer is prevented from setting higher health standards for employees than the secondary examining doctors choose to set."[19]

In *United Steelworkers of America* v. *Marshall*,[20] the D.C. Circuit reached the opposite result and upheld the multiple physician review procedure of the lead standard. According to the court, the provision is authorized by § 6(b)(7)'s broad mandate to require examinations that can "most effectively determine" a threat to worker health. In addition, the provision is reasonable in light of two findings supported by the record. First, lead diseases are often difficult to diagnose and multiple physician review increases the chances of a correct diagnosis. Second, some company physicians have engaged in the unsound and harmful practice of prophylactic chelation to reduce the blood-lead levels of employees. The court distinguished *Taylor*, where employees would seek multiple physician review to obtain a finding of fitness, thus forcing the employer to retain employees considered unfit by its own physician and standards. In the lead standard, the multiple physician review procedure was to prevent excess exposure of "leaded" employees and, together with the medical removal protection, the employer is not precluded from imposing more stringent health standards.

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[9] M. Rothstein, Medical Screening of Workers 88 (1984).

[10] 11 O.S.H. Cas. (BNA) 1464 (1983).

[11] 29 C.F.R. § 1910.1025(j)(4) (1984).

[12] Vinyl chloride, 29 C.F.R. § 1910.1017 App. A (1984); arsenic, 29 C.F.R. § 1910.1018 App. C (1984); lead, 29 C.F.R. § 1910.1025 App. C (1984); coke oven emissions, 29 C.F.R. § 1910.1029 App. B (1984); cotton dust, 29 C.F.R. § 1910.1043 Apps. B, C & D (1984); DBCP, 29 C.F.R. § 1910.1044 App. B (1984); acrylonitrile, 29 C.F.R. § 1910.1045 App. C (1984); ethylene oxide, 29 C.F.R. § 1910.1047 App. C (1984).

[13] 48 Fed. Reg. 17,315 (1983).

[14] 49 Fed. Reg. 25,734 (1984).

[15] B. Mintz, OSHA: History, Law, and Policy 136 (1984).

[16] Industrial Union Dept v. Hodgson, 499 F.2d 467, 485 (D.C. Cir. 1974).

[17] 599 F.2d 622 (5th Cir. 1979).

[18] 581 F.2d 493 (5th Cir. 1978), aff'd sub nom. Industrial Union Dept v. American Petrol. Inst., 448 U.S. 607 (1980).

[19] 599 F.2d at 625.

[20] 647 F.2d 1189 (D.C. Cir. 1980), cert. denied sub nom. Lead Indus. Assn., Inc. v. Donovan, 453, U.S. 913 (1981).

In the ethylene oxide standard, OSHA adopted the position taken by NIOSH that multiple physician review was unnecessary for ethylene oxide.

(6) Who pays for the examination?

Section 6(b)(7) of the act makes it clear that medical examinations shall be made available "by the employer or at his cost." OSHA's health standards have included language indicating that all costs for medical examinations must be borne by the employer. In *Phelps Dodge Corp.*,[21] the Commission held that a provision in the inorganic arsenic standard providing that medical examinations be provided without cost required the employer to compensate employees for time spent taking the examination (outside normal working hours) and for extra transportation expenses. The Commission's decision was affirmed by the Ninth Circuit.[22]

(7) What personnel action may be taken as a result of the examination?

With the exception of medical removal protection and rate retention under some health standards, OSHA has not indicated what personnel actions may or may not be based on OSHA-mandated medical surveillance. Consequently, unless there is an applicable provision in a collective bargaining agreement or the personnel action otherwise violates some antidiscrimination law (e.g., handicap laws), an employer may discharge, reassign, lay off, or refuse to hire employees on the basis of medical surveillance. The problem of job security is one major reason why employees sometimes do not fully cooperate with medical surveillance programs.[23]

Technical Note #C.2: OSHA Priorities in Risk Assessment and Risk Management

Section 6(b)(1) of the OSH Act directs the Secretary of Labor to promulgate standards "to serve the objectives of this act." Section 6(g) sets forth two criteria for standards development: the urgency of the need for the standard ("worst-first") and the recommendations from NIOSH.

In *National Congress of Hispanic American Citizens v. Marshall*, the D.C. Circuit reviewed OSHA's priorities for development of health and safety standards. For health standards, OSHA considers the number of workers exposed, the severity of the hazards, the existence of research relevant to hazard identification and methods of control, NIOSH recommendations, citizen petitions, court decisions, and other factors.[24] Using these criteria, OSHA generally has given its highest priority to carcinogenic substances.

Although White House priorities and congressional oversight and appropriations activity also affect standards promulgation, Congress has never spelled out its priorities for OSHA standards. According to OSHA's health standards chief, "the Federal agencies are not doing a competent job of regulating chemicals and part of the blame rests with Congress." In his view, there is a need for congressional guidelines in developing criteria for priorities for regulation, such as the nature of the hazard and the level of exposure.

OSHA has developed an internal document, RUL.1, which provides a framework for dealing with severity, exposure, risk, feasibility, and similar issues. According to a Reagan Administration official, the potency of the substance and the current exposure levels are two key factors in establishing the need to regulate a hazardous substance. A former DOL official asserted that although priority should be given to the gravest health hazards, OSHA cannot afford to use all of its resources here. Another OSHA official observed that OSHA is required by law to apportion its resources between reviewing old standards and developing new ones.

The difficult scientific and policy questions of deciding what substances should be regulated, in what order, and in what manner are further complicated by political considerations. Most observers probably would agree with the OSHA official who stated that "the setting of OSHA's priorities is, and always has been, highly politicized." A former OSHA chief under Ford and Carter commented that the priorities for standards-setting often depend on "who is making the most noise politically." In his view, this has been especially true during the Carter and Reagan Administrations. Another former OSHA chief and a current OSHA official contend that most of the pressure comes from the various interest groups rather than from the White House. Indeed, the degree of political pressure may be related to the type of regulation at issue. The former OSHA chief under Reagan stated that people are more reasonable in the safety area than in health. "Health issues involve politics at its lowest."

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[21] 11 O.S.H. Cas. (BNA) 1441 (1983), aff'd, 725 F.2d 1237 (9th Cir. 1984).

[22] Phelps Dodge Corp. v. OSHRC, 725 F.2d 1237 (9th Cir. 1984).

[23] M. Rothstein, Medical Screening of Workers 203-04 (1984).

[24] 626 F.2d 882 (D.C. Cir. 1979).

It is difficult to gauge the effects of such political maneuvering on the ultimate decisions of the agency. The former OSHA chief asserted that to be an effective head of OSHA "requires a strong-willed individual." Another former OSHA chief concurred that the "head of the agency must be strong enough to buck the political pressure."

Several of the individuals interviewed stated that political pressure is neither unanticipated nor totally undesirable. One official described attempts to influence OSHA policy as "helpful input." Another former OSHA chief under Reagan termed political pressure "a part of the game" and added that "it's the price we pay for having a free and open society." A former OSHA lawyer suggested that procedures established under the Act and the structure of our governmental system provide checks and balances on OSHA's decisionmaking.[25]

The way in which political considerations enter the decisionmaking process is also the cause of some concern. The director of OSHA's Office of Standards Review cautions that political pressure should influence only policy decisions, not the interpretation of scientific data. "If you don't want to regulate because of cost, say so. Don't prostitute the science."

Risk Assessment/Significant Risk

NIOSH Approach.—As a scientific and technical research agency, NIOSH approaches hazard control with the view of providing maximum protection for workers. Thus, although it need not determine whether a risk is "significant" in the legal sense, it does attempt to quantify the magnitude of risk. NIOSH's quantitative risk assessment attempts to identify hazards, assess exposure, evaluate possible dose-response relationships, and characterize risk. Unlike epidemiology, which is based solely on human data and observed levels of exposure, quantitative risk assessment considers data from animal studies as well and attempts to extrapolate risk estimates to lower levels than those observed in animals.

Because the courts require that OSHA standards contain increasingly detailed risk assessments, NIOSH has started more formal activities in quantitative risk assessment in the criteria documents division. A senior NIOSH epidemiologist who is leading this new effort says that NIOSH has little or no expertise in the field at present. Nevertheless, the agency is working with consultants to develop the capability to better quantify the need for standards. One of the goals of the new section is to develop working groups in various subject areas and, where needed, to use outside experts to assist with the risk assessments.

OSHA Approach.—Any discussion of risk assessment under OSHA necessarily begins with the Supreme Court's 1980 decision in the Benzene case. In *Industrial Union Department v. American Petroleum Institute* (API),[26] the Supreme Court addressed several important substantive issues in ruling on the validity of OSHA's benzene standard. The Fifth Circuit had invalidated the standard because OSHA failed to provide a quantitative estimate of the benefits to be achieved by reducing the permissible exposure limit (PEL) from 10 ppm to 1 ppm.[27]

The Fifth Circuit based its decision on § 3(8)'s definition of an "occupational safety and health standard" as being "reasonably necessary or appropriate" for safe workplaces. From this language the court held that the Secretary must determine "whether the benefits expected from the standard bear a reasonable relationship to the costs imposed by the standard."[28] The court was, essentially, fashioning a three-part test:

1. whether substantial evidence supports the Secretary's estimate of expected benefits;
2. whether substantial evidence supports the Secretary's estimate of expected costs; and
3. whether the benefits bear a reasonable relationship to the costs.

Because there was inadequate evidence of expected benefits, the other issues were not decided.

The Supreme Court affirmed the decision of the Fifth Circuit, but the Court was sharply divided, and issued five separate opinions. Justice Stevens, writing for a plurality of four justices, rejected the Government's argument that § 3(8) is meaningless and is supplanted by § 6(b)(5), which details the requirements for standards dealing with toxic materials or harmful physical agents. According to the plurality opinion, § 3(8) must be satisfied before there can be any consideration of a standard under § 6(b)(5).[29] "[Section 3(8)] requires the Secretary, before issuing any standard, to determine that it is reasonably necessary and appropriate to remedy a significant risk of material health impairment."[30] In other words, "the burden was on the Agency to show, on the basis of substantial evidence, that it is at least more likely than not that long-term exposure to 10 ppm of benzene presents a significant risk of material impairment."[31]

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[25] For a further discussion of how political factors influence OSHA, see D. McCaffrey, OSHA and the Policies of Health Regulation (1981).

[26] 448 U.S. 607 (1980).

[27] American Petrol. Inst. v. OSHA, 581 F.2d 493, 505 (5th Cir. 1978), aff'd, 448 U.S. 607 (1980).

[28] 581 F.2d at 503. The court relied on its prior construction, in Aqua Slide 'N' Dive Corp. v. Consumer Product Safety Commission, 569 F.2d 831 (5th Cir. 1978), of similar language in the Consumer Product Safety Act.

[29] Among the requirements of § 6(b)(5), a standard must be "feasible"

In effect, the plurality added a fourth element to the Fifth Circuit's test that had to be satisfied before the other three factors could even be considered. This "significant risk" requirement is not just an analytical starting point, it is an important substantive limitation on OSHA's rulemaking authority. According to the plurality, the Act "was not designed to require employers to provide absolutely risk-free workplaces," but was only intended to require "the elimination, as far as possible, of significant risks of harm."[32] The Fifth Circuit's decision was affirmed because the Secretary failed to prove that there are significant risks associated with benzene exposure at the present limits.

Justice Marshall's dissenting opinion accused the plurality of fashioning a restrictive rule of law from a definitional section of the statute that was not intended to have such a profound effect. The result is to place "the burden of medical uncertainty squarely on the shoulders of the American worker, the intended beneficiary of the Occupational Safety and Health Act."[33]

Significantly, of the two main points of the plurality opinion, the effect of § 3(8) and the sufficiency of the evidence supporting the need for a new standard, neither are majority views of the Court.[34] Justice Rehnquist, concurring in the judgment, joined the four dissenters in concluding that § 3(8) was not intended to be a general check on the Secretary's authority under § 6(b)(5).[35] As to the sufficiency of the evidence supporting the need for a new standard, Justice Rehnquist did not address this question and Justice Powell, in a separate concurrence,[36] conceded that the question was close. The four dissenters argued that the Secretary had presented sufficient evidence of the need for the standard.[37]

Courts applying the API tests to other cases challenging OSHA standards have reached different results. In *United Steelworkers of America* v. *Marshall*,[38] the D.C. Circuit, in upholding the validity of the lead standard, held that the Secretary had satisfied § 3(8)'s requirement of proving "significant harm." Instead of relying on "categorical assumptions" about lead poisoning, the Secretary amassed voluminous data of the harmful effects of lead at various blood-lead levels and correlated these levels with various average air-lead levels.

In *Texas Independent Ginners Association* v. *Marshall*,[39] however, the Fifth Circuit struck down the cotton gin standard, finding that the Secretary failed to prove that cotton dust in cotton gins poses a significant health risk . OSHA simply assumed that because byssinosis results from high exposure levels in textile mills that byssinosis also results from the lower exposure levels in cotton gins. This assumption did not satisfy the § 3(8) requirement of significant harm, especially in light of the seasonal nature of cotton gin operations.

An important part of risk assessment and "significant risk" is the quality of the scientific data on which the risk assessment is based. Section 6(b)(5) of the Act provides that standards dealing with toxic materials or harmful physical agents must be based on the "best available evidence." While this language appears to be straightforward, the scientific evidence of the precise harmful effects of exposure to various substances is often inadequate, incomplete, inconclusive, or subject to dispute. At the same time, there may be clear evidence that exposure at some levels to these substances causes serious illness.[40] This dilemma has raised two related questions in the context of § 6(b)(5): 1) What constitutes the "best available evidence?" and 2) Is OSHA precluded from adopting new standards until there is definitive, detailed, and indisputable scientific evidence?

In the *Benzene* case, the Secretary argued that because there is no absolutely safe level known for benzene, industry should have the burden of showing that a safe level exists. Any other approach, it was argued, would require OSHA to wait for deaths to occur before taking any action.

The plurality opinion specifically rejected this argument and, as discussed previously, held that OSHA had the burden of proving that it is at least more likely than not that long-term exposure to benzene at the present PEL presents a significant risk of material health impairment. According to the plurality, this burden will not prevent OSHA from regulating carcinogens for the following reasons. First, it is OSHA's responsibility to determine, in the first instance, what it considers to be a significant risk. Although there is no duty to calculate the exact probability of harm, OSHA does have the obligation to determine whether a significant risk is present. Second, a standard need not be based on scientific certainty, and OSHA is free to risk error on the side of over-protection so long as the standard is supported by a body of reputable scientific thought. Third, the relative significance of risk can be quantified in a number of ways other than by epidemiological studies, such as by extrapolation of animal test data.[41]

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[30] 448 U.S. at 639. The court incorrectly paraphrased § 3(8) as requiring a standard to be "reasonably necessary and appropriate." Actually, a standard need only be "reasonably necessary or appropriate."

[31] 448 U.S. at 653.

[32] Id.

[33] 448 U.S. at 690.

[34] See generally Note, Plurality Decisions and Judicial Decisionmaking, 94 Harv. L. Rev. 1127 (1981).

[35] 448 U.S. at 681. In his view, § 6(b)(5) was too vague and therefore represented an unconstitutional delegation of legislative authority to the executive.

[36] 448 U.S. at 667. Justice Powell believed that the Secretary failed to prove the economic feasibility of the standard.

[37] For criticism of the Court's decision, see M. Rothstein, Occupational Safety and Health Law 73-74 (2d ed. 1983); Rodgers, Judicial Review of Risk Benzene Decision, 11 Envtl. L. 301 (1981).

[38] 647 F.2d 1189 (D.C. Cir. 1980), cert. denied sub nom. Lead Indus. Assn., Inc. v. Donovan, 453 U.S. 913 (1981).

[39] 630 F.2d 398 (5th Cir. 1980).

[40] See generally McGarity, Substantive and Procedural Discretion in Administrative Resolution of Science Policy Questions: Regulating Carcinogens in EPA and OSHA, 67 Geo. L.J. 729 (1979).

In *Texas Independent Ginners Association* v. *Marshall*,[42] the Fifth Circuit held that the cotton gin standard was not based on the best available evidence. OSHA had based the standard on studies of ginning employees in Egypt, Uganda, Greece, and Sudan, rather than on a study of American gins, where there is reduced exposure due to the seasonal nature of the work. OSHA also overrelied on studies of byssinosis in the cotton manufacturing industry. Finally, OSHA failed to reopen the hearing record to consider a more recent study. On this final point, it is not clear what the practical limits should be for imposing an ongoing duty on OSHA to consider new evidence, inasmuch as new scientific information is being discovered on a continuing basis.

The *Benzene* decision has certainly caused OSHA to reevaluate the way in which scientific research is translated into regulatory action. Nevertheless, it has not viewed the decision as an insurmountable barrier, according to former Carter and Reagan OSHA Chiefs.

After the *Benzene* decision, the arsenic standard, which was pending before the Ninth Circuit, was remanded to OSHA for the completion of a risk assessment. In January 1983, OSHA published its final risk assessment for arsenic and in so doing set forth its general framework for evaluating the need for a standard.[43] In setting health standards OSHA uses a four-step approach:

1. Risk assessments are performed where possible and are considered with other relevant factors to determine whether the substance to be regulated poses a significant risk to workers.
2. OSHA considers which, if any, of the proposed standards being considered for that substance will substantially reduce the risk.
3. OSHA looks at the best available data to set the most protective exposure limit necessary to reduce significant risk that is both technologically and economically feasible.
4. OSHA considers the most cost-effective way to achieve the objective.

Risk assessment, therefore, is the first step in the process of regulation. OSHA defines quantitative risk assessment as "an attempt to predict the degree of risk associated with a specific level of exposure. This is done either through direct observation or by extrapolation. . . ." Some important components of risk assessment are a description of the hazard, a description of the potential exposure and worker scenarios, a description of the dose-response relationship, and a quantitative determination of risk.[44]

According to some published reports, there is a danger in over-reliance on quantitative risk assessment. To begin with, the ability to generate detailed and precise mathematical models for hazards varies greatly. To require both detail and precision may be either impossible or so time-consuming that no action is taken on hazards clearly in need of regulatory action. (The court's recent decision on the asbestos ETS is an example). Thus, it has been argued that underlying policy questions should be addressed even without detailed quantitative models.[45]

Second, "risk assessment" should not be confused with "risk management," the latter being the process of evaluating alternative regulatory actions and selecting among them.[46] Risk assessment, quantitative or qualitative, cannot substitute for the value judgments and policy review essential to regulation. Administrative actions do not automatically result from risk assessment.

Third, there is a lack of uniformity in the way that Federal agencies conduct risk assessment.[47] Although there is widespread support for the use of a single methodology and interagency cooperation, there is disagreement about whether a single agency is needed to perform risk assessments.

Risk Acceptability

In the *Benzene* case, the Supreme Court's plurality opinion stated:

> [T]he [OSHA] statute was not designed to require employers to provide absolutely risk-free workplaces whenever it is technologically feasible to do so. . . There are many activities that we engage in every

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[41] 448 U.S. 607, 656-58 (1980).

[42] 630 F.2d 398 (5th Cir. 1980).

[43] 48 Fed. Reg. 1864 (1983).

[44] See generally Congressional Research Service, A Review of Risk Assessment Methodologies, House Subcommittee on Science, Research, and Technology, 98th Cong., 1st sess. (Comm. Print 1983).

[45] See Cranor, Epidemiology and Procedural Protections for Workplace Health in the Aftermath of the Benzene Case, 5 Indus. Rel. L.J. 372 (1983).

[46] National Academy of Sciences, Risk Assessment in the Federal Government: Managing the Process 18 (1983).

[47] Id. at 4-7.

> day—such as driving a car or even breathing city air—that entail some risk of accident or material health impairment; nevertheless, few people would consider these activities "unsafe." Similarly, a workplace can hardly be considered "unsafe" unless it threatens the workers with a significant risk of harm.[48]

The above quote indicates that only significant risks are appropriate for regulation and that the presence of some degree of risk in the workplace is inevitable and thus "acceptable."[49]

It is difficult to define or quantify what is an acceptable risk. The acceptability of a risk depends on the nature of the risk, its severity, the level of exposure, the economic consequences, the alternatives, the views of the decisionmaker and, perhaps most importantly, the question of acceptable to whom? It may be that it is unnecessary to define what risks are acceptable, at least in the regulatory context. OSHA has not said what is acceptable, only what is not.

The specific question of what is an acceptable risk under OSHA has, apparently, not yet materialized. Two OSHA officials indicated that because of recent court decisions, OSHA must be particularly concerned about the issues of significant risk and feasibility before it takes regulatory action. A third added, however, that the notion of acceptable risk is "inherent in decisionmaking" and that it influences decisions in practical ways.

There is also some concern that the concept of "acceptable risk" is used by those who argue, in effect, that some jobs are inherently unsafe or unhealthful and that the government is misguided in its attempts to eliminate all of these risks. A Carter OSHA Chief charged that "acceptable risk" is a "non-issue in OSHA. Nobody is suggesting that it is possible to have zero risk in the workplace." Moreover, she asserted that this "phony issue" is raised by those individuals opposed to any regulation of the workplace.

Technical Note #C.3: Technological Feasibility of OSHA Health Standards

The issue of technological feasibility could arise if OSHA attempted to require the use of engineering controls to reduce exposure to levels that would not be harmful to the reproductive health of any workers or their offspring. Because of evidence suggesting that extremely low levels of exposure could be harmful, it might be asserted that it is technologically infeasible to achieve the required reductions in exposure levels.

The congressional purpose of the OSH Act, to assure safe and healthful workplaces, is qualified by the phrase "so far as possible." This language indicates that the Secretary must promulgate standards that are technologically achievable. Even before a standard is proposed, OSHA considers whether it is feasible, and in so doing may modify an "absolute" standard recommended by NIOSH or another body. Nevertheless, a standard may be promulgated that contemplates future improvements in safety and health technology.

Section 6(b)(5), which applies to new standards regulating toxic substances or harmful physical agents, contains two references to the requirement of feasibility. First, in promulgating standards under § 6(b)(5), the Secretary "shall set the standard which most adequately assures, to the extent feasible . . . that no employee will suffer material impairment of health. . . ." Second, in addition to the attainment of the highest degree of protection for employees, "other considerations shall be . . . the feasibility of the standards . . . ."

In *Society of the Plastics Industry, Inc.* v. *OSHA*,[50] the Second Circuit indicated that a defense based on technological infeasibility requires showing that a standard is "clearly impossible of attainment." The court stated that OSHA may require improvements in existing technologies or the development of new technologies.[51]

Similar reasoning was used by the Third Circuit in *AFL-CIO* v. *Brennan*,[52] although it reached the opposite result. In ruling on the feasibility of a mechanical power press standard, the court declared that "at least to a limited extent, OSHA is to be viewed as a technology-forcing piece of legislation."[53] Nevertheless, the court found that compliance with the standard was not technologically feasible "in the near future."[54]

Decisions of the courts of appeals have attempted to clarify this "technology-forcing" language. In *American Iron & Steel Institute* v. *OSHA*,[55] the Third Circuit indicated that even though the Secretary may require an employer "to implement technology 'looming on today's horizon,' . . . the statute does not permit the Secretary to place an affirmative duty on each employer to research and develop new technology."[56] According to the court, this is especially true when the research and development provisions are speculative and render any assessment of feasibility practically impossible.

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[48] Industrial Union Dept. v. American Petrol. Inst., 448 U.S. 607, 641-42 (1980).

[49] See Dinman, Occupational Health and the Reality of Risk—An Eternal Dilemma of Tragic Choices, 22 J. Occup. Med. 153 (1980).

[50] 509 F.2d 1301 (2d Cir. 1975). See generally Doniger, Federal Regulation of Vinyl Chloride: A Short Course in the Law and Policy of Toxic Substances Control, 7 Ecology L.Q. 497 (1978).

[51] 509 F.2d at 1309.

[52] 530 F.2d 109 (3d Cir. 1975).

[53] Id. at 121 (footnote omitted).

[54] Id. at 122. See Industrial Union Dept. v. Hodgson, 499 F.2d 467, 479-80 (D.C. Cir. 1974).

[55] 577 F.2d 825 (3d Cir. 1978), cert. dismissed sub nom. Republic Steel Corp. v. OSHA, 448 U.S. 917 (1980)(coke oven emissions standard).

[56] 577 F.2d at 838. See 47 Cin. L. Rev. 477 (1978).

In *United Steelworkers of America* v. *Marshall*,[57] the D.C. Circuit delineated OSHA's burden of proving technological feasibility. "OSHA's duty is to show that modern technology has at least conceived some industrial strategies or devices which are likely to be capable of meeting the PEL and which the industries are generally capable of adopting."[58] The court's limited role in deciding whether this burden has been met was set out in the D.C. Circuit's opinion in *AFL-CIO* v. *Marshall*:[59]

> Judging the technological feasibility of a particular agency goal is beyond the expertise of the judiciary especially where the assessment involves predictions of technological changes. Instead, our task on review is to find whether the agency sufficiently supported its feasibility determination with material in the record.[60]

Economic Feasibility.—A related argument that is likely to be raised is that it is economically infeasible to reduce exposures to the levels where no harms would occur.

In *American Textile Manufacturers Institute, Inc.* v. *Donovan*, [61] the Supreme Court addressed the issue of whether the Act requires the Secretary, in promulgating a standard under § 6(b)(5), to determine that the costs of the standard bear a reasonable relationship to its benefits. The Fifth Circuit, in the *Benzene* case,[62] had imposed such a requirement. The D.C. Circuit, however, in the cotton dust[63] and lead[64] cases had rejected this view.[65]

In a five-to-three decision,[66] the Court rejected the argument that the Act requires the use of cost-benefit analysis. Relying on the plain meaning of the word "feasible" as "capable of being done," the Court ruled that imposing a cost-benefit requirement would be inconsistent with the mandate of Congress:

> Congress itself defined the basic relationship between costs and benefits, by placing the "benefit" of worker health above all other considerations save those making attainment of this "benefit" unachievable. . . . Thus, cost-benefit analysis by OSHA is not required by the statute because feasibility analysis is.[67]

The Court observed that when Congress has intended that an agency engage in cost-benefit analysis, it has clearly indicated such an intent on the face of the statute.[68] Neither the language of OSHA nor its legislative history indicate such a congressional intent.

According to the majority opinion of Justice Brennan, "feasible" as used in § 6(b)(5) includes economic feasibility. After reviewing the record, the Court concluded that the D.C. Circuit did not err in holding that the Secretary's finding that compliance with the cotton dust standard was economically feasible was supported by substantial evidence. Even though no specific economic studies were performed on the final standard, there were studies that showed that compliance with a stricter and more costly standard was feasible.[69]

Two further points relative to the *ATMI* case are worthy of mention. First, the holding is limited to § 6(b)(5) standards; the Court did not address the issue of whether cost-benefit analysis is required in promulgating other types of standards.[70] Second, despite assertions to the contrary,[71] the Secretary is not even permitted to engage in cost-benefit analysis in promulgating standards pursuant to § 6(b)(5). Besides feasibility analysis, "Congress did not contemplate any further balancing by the agency for toxic material and harmful physical agents standards. . . ."[72]

After the cotton dust decision, OSHA indicated that it would not engage in cost-benefit analysis, but that it would use cost-effectiveness analysis. While the former would consider whether the benefits of a regulation are sufficient to outweigh its costs, the latter is concerned with the most efficient way of attaining a certain level of protection.

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[57] 647 F.2d 1189 (D.C. Cir. 1980), cert. denied sub nom. Lead Indus. Assn., Inc. v. Donovan, 453 U.S. 913 (1981).

[58] 647 F.2d 1189.

[59] 617 F.2d 636 (D.C. Cir. 1979), aff'd sub nom. American Textile Mfrs. Inst. v. Donovan, 452 U.S. 490 (1981)(cotton dust standard).

[60] 617 F.2d at 656.

[61] 452 U.S. 490 (1981).

[62] American Petrol. Inst. v. OSHA, 581 F.2d 493, 503 (5th Cir. 1978), aff'd on other grounds sub nom. Industrial Union Dept. v. American Petrol. Inst., 448 U.S. 607 (1980).

[63] AFL-CIO v. Marshall, 617 F.2d 636, 664 (D.C. Cir. 1979), aff'd sub nom American Textile Mfrs. Assn., Inc. v. Donovan, 452 U.S. 490 (1981).

[64] United Steelworkers of America v. Marshall, 647 F.2d 1189 (D.C. Cir. 1980), cert. denied sub nom. Lead Indus. Assn., Inc. v. Donovan, 453 U.S. 913 (1981).

[65] See also American Iron & Steel Inst. v. OSHA, 577 F.2d 825, 836 (3d Cir. 1978) (upholding validity of coke oven emissions standard despite an annual compliance cost of $240 million), cert. dismissed sub nom. Republic Steel Corp. v. OSHA, 448 U.S. 917 (1980).

[66] Justice Powell took no part in the decision, but in his concurring opinion in the API case, he indicated that he would require cost-benefit analysis. Thus, as to this issue, it would appear that the Court is divided five-to-four. Justice Stewart, since replaced by Justice O'Connor, voted with the dissent in ATMI.

[67] 452 U.S. at 509 (footnote omitted).

[68] Id. at 510.

[69] Id. at 522-36.

[70] Id. at 509 n.29.

[71] Id. at 544 (Rehnquist, J., dissenting).

[72] Id. at 513.

Technical Note #C.4: Coverage of Employees, Employers, and Chemicals Under The Hazard Communication Standard

Employees

Of the 25 million workers "potentially exposed" to chemical health hazards, approximately 15 million are covered by the hazard communication standard.[73] OSHA's standard applies only to manufacturers, importers, and distributors of "hazardous chemicals," and is limited to firms in certain standard industrial classification (SIC) codes.[74] Furthermore, the standard does not cover nonmanufacturing personnel (e.g., office workers), though they may work in the chemical manufacturing sector and may be exposed to toxic chemicals. Also unprotected by the regulation are commercial, nonmanufacturing "downstream" users of chemicals, agricultural workers, and public employees. OSHA's rationale for limiting coverage to selected employees of chemical manufacturers in SIC codes 20-39 results from the desire to create a cost-effective rule. The agency based the regulation on data indicating that half of "chemical source" illnesses and injuries occur at these worksites.[75]

Most State "right-to-know" laws provide coverage to a larger worker population than does OSHA. The laws are seldom limited to persons engaged in the manufacture of hazardous chemicals, and most include all employees who will come in contact with hazardous chemicals at the workplace, though domestic workers are expressly excluded from coverage in several States. Furthermore, several State laws expressly cover public employees.

Employers

OSHA's standard applies to a selective, albeit large, portion of chemical manufacturers and importers.[76] To avoid interagency jurisdictional disputes, OSHA has exempted from coverage pesticides and hazardous wastes (subject to EPA regulations), food additives (regulated by FDA), distilled spirits (controlled by BATF), and consumer products (subject to CPSC regulations).[77] Similarly, miners are exempt due to coverage by the Mine Safety and Health Act.

As discussed earlier, most State "right-to-know" laws cover all employees exposed to chemical hazards in the workplace. Some States, however, such as West Virginia, exclude certain industries.[78]

Hazardous Chemicals

OSHA's hazard communication standard requires chemical manufacturers and importers to assess the hazards of chemicals they produce or import to which workers may be exposed.[79] Certain chemicals are not subject to this requirement, and chemicals produced and used in laboratories are subject to less stringent regulations. Information about chemicals determined to be hazardous must be communicated to workers.

Chemical exposures which result in acute or chronic health effects are considered health hazards.[80] The determination of hazardness is to be based on "evidence that is statistically significant and that is based on at least one positive study conducted in accordance with established scientific principles."[81] In determining whether a chemical poses a health hazard, an employer may consult a list of "available data sources" provided by OSHA.[82] It is important to recognize, however, that use of these sources is advisory, not mandatory. One of the chief criticisms of the OSHA regulation is the advisory nature of the source lists. Some argue that employers are granted too much discretion in determining whether a chemical poses a hazard.[83] And some observers contend that employers may not report all that is known about a chemical's hazards.[84]

Another criticism of the regulation concerns the concentration levels established by OSHA. The standard requires disclosure of substances that contain 0.1 percent (or more) of carcinogens, or that contain 1 percent (or more) of chemicals otherwise identified as hazardous.[85] Critics maintain that the concentration levels set by OSHA are arbitrary, and do not provide adequate safeguards to protect worker health.[86]

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[73] See Preamble to final OSHA Standard on Workplace Hazard Communication, O.S.H. Rep. (BNA) 700, 748 (Dec. 1, 1983); Workers' "Right-to-Know": OSHA's Hazard Communication Rule, Issue Brief IB84103, The Library of Congress Congressional Research Service 2 (1984).

[74] 29 C.F.R. § 1910.1200(b) (1984). OSHA officials have, since the promulgation of the Hazard Communication Standard, undertaken a review of the limited coverage of the regulation. At the present time, OSHA is considering expanding the scope of the standard to include all employers. OSH Rep. (BNA) 761, 763 (Mar. 7, 1985).

[75] See Preamble to final OSHA Standard on Workplace Hazard Communication, O.S.H. Rep. (BNA) 700, 748 (Dec. 1, 1983).

[76] Id. at 705-07.

[77] 29 C.F.R. §§ 1910.1200(b)(4), (5) (1984).

[78] W. Va. Code § 21-3-18(c) (1981).

[79] 29 C.F.R. § 1910.1200(b)(3), (d)(i) (1984).

[80] Id. at § 1910.1200(c).

[81] Id. at § 1910.1200(d)(2).

[82] Id. at § 1910.1200 (app. C).

[83] Workers' "Right-to-Know": OSHA's Hazard Communication Rule, Issue Brief IB84103, The Library of Congress Congressional Research Service 2, 4 (1984).

[84] Id.

[85] 29 C.F.R. § 1910.1200(d)(5).

[86] Workers' "Right-to-Know": OSHA's Hazard Communication Rule, Issue Brief IB84103, The Library of Congress Congressional Research Service 2, 3 (1984).

States regulating this area have taken an active role in determining the substances for which an employer must provide information to workers. Most States (rather than the manufacturers) determine which chemicals are subject to their right-to-know laws. This ensures a greater likelihood of compliance with the statutory requirements (by removing uncertainty as to the substances regulated), and provides for enhanced effectiveness in reaching statutory goals.

Furthermore, many States define a "hazardous" chemical more broadly than does OSHA. New Jersey, for example, lists nearly 2,000 substances as "hazardous" chemicals.[87]

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[87] Chemical Right-to-Know Requirements: Federal and State Laws and Regulations on Disclosure, Special Report (BNA) 3 (1984).

Appendix D

Technical Notes: EPA

Technical Note #D.1: Information Sources Under TSCA

Section 4: Testing Rules[1]

Section 4 of TSCA may be of great importance in developing information about a range of reproductive health hazards. It directs EPA to promulgate testing rules to develop data with respect to health effects of existing or new chemicals if a chemical may present an unreasonable risk of injury to health or the environment, is produced in substantial quantities and may reasonably be anticipated to enter the environment in substantial quantities, or may cause significant or substantial human exposure.

In such a testing rule, EPA can prescribe standards for the development of data by chemical manufacturers on mutagenicity, teratogenicity, behavioral disorders, and any other effects.[2]

To date, the only testing rule that has been finalized is for 1,1,-trichloroethane, which includes protocols for the development of data on fetal defects and abnormal development. Several other rules have been proposed.[3]

Critics of § 4 claim that administrative delays and the inability of testing protocols to be designed through regulatory rulemakings have made § 4 unworkable.[4] This criticism appears valid since scientific consensus on the types of studies needed and their specific design are difficult to reach through formal rulemakings. In response to these problems, EPA began to negotiate voluntary testing agreements for several chemicals for which the agency has made informal findings of an unreasonable risk. Under these negotiated testing protocols (which rely to a certain extent on testing screens), laboratory and subclinical testing of reproductive health hazards can be emphasized just as in § 4 testing rules. In July 1984, however, a Federal trial court ruled that such voluntary testing agreements were illegal.[5]

One related issue is whether data reported to EPA under these testing agreements can be obtained by the public. Health data generated under testing rules are not subject to confidentiality claims by the manufacturer of an existing or new chemical under TSCA.[6] Therefore, information on reproductive health hazards can be obtained by the public.

Testing data reported under § 4 can also be used to provide a basis for regulatory action under other parts of TSCA to ban or control the production, use, or method of disposal of chemicals. Section 4(f) of the Act may be particularly important because it provides the basis for expedited agency regulatory review of substances suspected on the basis of testing or other data accumulated by the agency to pose a significant risk.

Under § 4(f), if EPA receives test data or any other information "which indicates to the Administrator that there may be a reasonable basis to conclude that a chemical substance or mixture presents or will present a significant risk of serious or widespread harm to human beings from *cancer, gene mutations or birth defects*, the Administrator shall 'initiate appropriate action under §§ 5, 6, or 7 to prevent or reduce to a sufficient extent such risk or publish in the Federal Register a finding that such risk is not unreasonable' "[7] (emphasis added). Section 9 of the Act requires EPA to report findings under § 4(f) to OSHA for appropriate action, but does not limit EPA's ability to act itself.[8] (See discussion of § 9 below.) Should EPA publish findings under § 4(f) that the risks of a substance are not unreasonable, those findings can be challenged in court.[9]

Section 4(e) of TSCA also directs EPA to establish an Interagency Testing Committee (ITC), to include members appointed by the Secretary of Labor and the Director of the National Institute of Occupational Safety and Health. The purpose of the ITC is to establish a list of chemical substances requiring testing rules under § 4(a). The Committee is directed to give priority to those substances "which are known to cause or contribute to or which are suspected of causing cancer, gene mutations, or birth defects."[10] EPA must publish a testing rule within 12 months of the listing of a substance by the ITC.[11]

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[1] 15 U.S.C. § 2603 (1982).

[2] Before prescribing epidemiological studies of workers in these testing rules, however, the Administrator must consult with the Director of NIOSH. 15 U.S.C. § 2603(b)(2)(A)(1982).

[3] 49 Fed. Reg. 39,810 (1984).

[4] See U.S. EPA (OTS), Priorities and Progress, 28-29 (July 1983). GAO has also endorsed negotiated testing agreements as "reasonable." GAO, EPA Implementation of Selected Aspects of the Toxic Substances Control Act, Dec 7, 1982 (GAO/RCED-83-62).

[5] NRDC and Industrial Union Department v. Ruckleshaus, No. 83-8844 (S.D.N.Y. Aug. 23, 1984) (Court order voids 16 testing agreements).

[6] 15 U.S.C. § 2613(b) (1982).

[7] 15 U.S.C. § 2603(f) (1982).

[8] 15 U.S.C. § 2608(a) (1982).

[9] 15 U.S.C. § 2618 (1982). These rules were used, for instance, to challenge EPA's decision in early 1982 not to list formaldehyde under § 4(f). NRDC v. Ruckleshaus, No. 83-2034 (D.C. Cir. filed July 18, 1983). The agency subsequently published an advanced notice of proposed rulemaking for formaldehyde, 48 Fed. Reg. 52,507 (1983).

[10] 15 U.S.C. § 2603(e) (1982).

[11] 15 U.S.C. § 2603(e)(1)(A)(viii) (1982).

Section 5: New Chemicals[12]

Section 5(a)(1) prohibits the manufacture of a new chemical without notification to EPA.[13] This provides another means for screening chemical substances for reproductive toxicity before the chemicals are manufactured commercially, since premanufacture notification (PMN) must be accompanied by a minimum set of health and environmental exposure and production data at least 90 days before the manufacture or processing of the substance begins. Unfortunately, according to studies prepared by OTA[14] and the General Accounting Office,[15] fewer than 50 percent of all PMNs that EPA receives include toxicity data and only about 20 percent of these contain information about a chemical's mutagenicity. Most critics assert that this is because EPA's PMN regulations allow manufacturers to avoid the submission of these kinds of data.[16]

EPA's review of PMNs involves an assessment of risks for each chemical based on a substance's toxicity and the nature and extent of human exposure, including occupational and environmental exposure. Health and exposure data in PMNs, subject to certain types of confidentiality claims, are available for public examination by interested persons.[17]

There are several ways in which EPA can regulate the production of a substance for which there may be human health hazards, but when there are insufficient data available to ban the chemical's production under the Act's regulatory mechanisms. Under § 5(a)(2), EPA may determine that certain future uses or exposures of a chemical or class of substances will constitute a "significant new use" for which the manufacturer must file a PMN under § 5(a)(1).[18] Such a determination can be made by publishing a significant new use rule (SNUR), so that if production volume, route of exposure, or use of the substance changes, a PMN including new exposure and production data must be submitted before the new use is authorized. Under § 5(e),[19] EPA may also issue a proposed order that limits production, distribution, and use of certain substances if the agency determines that insufficient information has been generated to evaluate the risk to human health or the environment. EPA can also promulgate a testing rule under §4, discussed above, in order to develop health and exposure data about a chemical or a particular use.

Section 5(f)[20] is also important with respect to potential reproductive health hazards. It provides that the Administrator can take immediate action to protect the public's health and welfare on the basis of information received through a PMN that the manufacture, processing, distribution in commerce, use, or disposal of a chemical substance presents or will present an unreasonable risk of injury to human health. The Administrator may propose an administrative order to limit the amount of the substance that can be manufactured, processed, or distributed in commerce, or he may petition a U.S. District Court to prohibit the manufacture, processing, or distribution in commerce until a regulatory action can be completed under § 6 of the Act.

Section 8: Reporting and Recordkeeping Requirements[21]

This section enables EPA to acquire valuable information concerning significant adverse health reactions and other important exposure and health effects information about new and existing chemicals. Because health effects information cannot be claimed as confidential by manufacturers under TSCA's provisions,[22] §8 can also provide valuable information to workers who suspect they have been exposed to hazardous substances, including information about other workers exposed to similar substances or mixtures in their employment. While small businesses are generally exempt from § 8's reporting requirements,[23] they may also be required by EPA to maintain and submit reports concerning chemicals for which testing or regulatory actions are pending.[24] The following sections detail these provisions.

Recordkeeping.[25]—Section 8(a) authorizes EPA to require manufacturers of existing chemicals (i.e., those not subject to PMN requirements) to maintain records or submit reports on information that is "known to or reasonably ascertainable" to the extent this information is necessary to administer TSCA. Section 8(a) must be implemented by rulemaking for specific chemicals or classes of substances. Through the use of this provision, EPA can accumulate information about substances that are suspected of having reproductive effects associated with their manufacture, processing, use, disposal, or byproducts. The section also specifies that the Administrator may require estimates of the number of people exposed to a substance in the workplace.[26]

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[12] 15 U.S.C. § 2604.

[13] 15 U.S.C. § 2604(a)(1).

[14] U.S. Congress, Office of Technology Assessment, The Information Content of Premanufacture Notices, 49-53 (OTA-BP-H-17, 1983).

[15] See, e.g., GAO, EPA Implementation of Selected Aspects of the Toxic Substances Control Act, Dec. 7, 1982 (GAO/RCED-83-62).

[16] For instance, see exemptions allowed under 15 U.S.C. § 2604(h) (1982). See also 47 Fed. Reg. 33,896 (1982) (site-limited and low volume chemicals intermediates); 47 Fed. Reg. 33, 896 (1982) (site-limited and low volume chemicals intermediates); 47 Fed. Reg. 33,924 (1982).

[17] See 15 U.S.C. §§ 2604(H), 2613(b).

[18] 15 U.S.C. § 2604(a)(1).

[19] 15 U.S.C. § 2604(e).

[20] 15 U.S.C. § 2604(f).

[21] 15 U.S.C. § 2607.

[22] 15 U.S.C. § 2613(b).

[23] 15 U.S.C. § 2607(a)(3)(A). See 40 C.F.R. § 710.2(X) (defining small manufacturer).

[24] 49 Fed. Reg. 45,425 (codified at 40 C.F.R. pt. 704 (1984)).

[25] 15 U.S.C. § 2607(a).

EPA published final general information reporting rules and a final information assessment rule in June 1982.[27] The rules cover 250 chemicals, as opposed to the 2,226 substances listed in the earlier 1980 rules implementing this section for obtaining general information on these chemicals.[28] Additional chemicals have been designated for reporting under § 8(a).[29] In June 1983, EPA published a methodology for releasing data not subject to confidentiality protections it has received pursuant to § 8(a).[30]

In addition to promulgating general reporting rules, EPA has used its authority under § 8(a) to require reporting on specific chemicals. In 1980 it issued a rule requiring reporting of the manufacture or proposed manufacture or import of Tris (2, 3-dibromopropyl), phosphate, and polybrominated biphenyls (PBBs).[31] Final asbestos reporting rules were issued in July 1982.[32] The agency proposed reporting requirements for chlorinated terphenyls in April 1983.[33]

Inventory.—TSCA § 8(b) requires EPA to compile and maintain an inventory of chemicals in production and distributed in commerce. This inventory is to be regularly updated and can provide some structural activity information about chemicals that are suspected reproductive health hazards. Final reporting regulations for the submission of data for the compilation of the § 8(b) inventory were issued in December 1977.[34]

Substances not listed in the inventory are subject to premanufacturing notice requirements under § 5. Amended twice, the most recent supplement of the inventory was published in May 1982. Section 8(b) also requires persons who manufacture chemicals or mixtures solely for scientific experimentation to maintain and submit records on these chemicals' production volume and worker exposure to EPA.[35]

Significant Adverse Reactions.—Section 8(c) requires chemical manufacturers and processors to maintain records of "significant adverse reactions to health or the environment, as determined by the Administrator by rule, alleged to have been caused by the substance or mixture." Significant adverse reactions are reactions that may indicate a tendency of a chemical or mixture to cause *long-lasting or irreversible damage* to health or the environment.[36] This may not therefore include temporary illnesses such as nausea or headaches, but would probably include sterility, albeit temporary, although this is not clearly indicated in the regulation.[37] Section 8(c) requires companies to keep all employee allegations deemed by the company to be significant adverse reactions for 30 years and all other allegations for 5 years. These records, if obtainable from companies, may provide valuable information to substantiate effects for certain occupational uses of chemicals. EPA published final rules implementing § 8(c) in August 1983.[38]

There are several important limiting factors on the use of this rule. "Already known human effects" discussed in medical and scientific literature do not have to be reported.[39] All manufacturers and many processors are subject to the regulation, but distributors and retailers who do not manufacture or process chemicals are not. The rule contains no automatic reporting requirements once a notice is submitted, but EPA has stated that it may require reporting at a later time. (The proposed rule had required automatic reporting of allegations if three similar allegations were recorded within 1 year for a particular substance.)[40] Thus, obtaining such reports may be limited, except when they are clearly identifiable and can be obtained by discovery in tort litigation.

Health and Safety Studies Reporting.—Section 8(d) of TSCA may also be a significant source of information about chemicals that are suspected of causing reproductive effects in occupational settings. It directs the Administrator to promulgate rules requiring chemical manufacturers and processors to submit to EPA copies of safety and health studies conducted by companies.[41]

The term "health and safety" study is defined by TSCA as:

> . . . any study (including laboratory studies) of any effect of any chemical substance or mixture on health or the environment or on both, including underlying data and epidemiological studies, studies of occupational exposure to a chemical substance or mixture, and any test performed pursuant to this act.[42]

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[26] 15 U.S.C. § 2607(a)(2)(F).

[27] 47 Fed. Reg. 26,992 (1982).

[28] 45 Fed. Reg. 13,646 (1980) (used to obtain general exposure data on 2,226 chemicals).

[29] 48 Fed. Reg. 22,697 (1983).

[30] 48 Fed. Reg. 27,041 (1983).

[31] 45 Fed. Reg. 70,728 (1980).

[32] 46 Fed. Reg. 70,728 (1982) (recodified at 48 Fed. Reg. 23,420 (1983)).

[33] 47 Fed. Reg. 33,298 (1982).

[34] 48 Fed. Reg. 19,419 (1983).

[35] 42 Fed. Reg. 64,572 (1977). See Bronstein and Nunverberg, Section 8(b) of the Toxic Substances Act: A Case Study of Government Regulation of the Chemical Industry, 13 Nat. Resources L.J. 706 (1981).

[36] 15 U.S.C. § 2607c.

[37] Environmental Law Institute, EPA Authority and Activities Relating to Occupational Reproductive Hazards (1984) (unpublished report).

[38] 48 Fed. Reg. 38,178 (1983).

[39] Id.

[40] 45 Fed. Reg. 47,008 (1980).

[41] The term "processor" may cover "end-user" but this is not clear under the statute, and may be thrown into doubt because in other parts of the statute the word "users" is employed. However, as discussed later, EPA's regulations would cover anyone in possession of such studies.

[42] Proposed rule at 45 Fed. Reg. 47,008 (1980).

Section 8(d) includes two sets of requirements. First, under subpart 8(d)(1), manufacturers and processors must submit lists of health and safety studies conducted by them, known to them, or reasonably ascertainable to them. Second, subpart 8(d)(2) requires those in possession of a study to submit copies of any study contained on the list or otherwise known to the person. EPA first promulgated regulations implementing §8(d) in 1978 that required reporting of studies on chemicals listed in the first Interagency Testing Committee report.[43] The rule was challenged in the Third Circuit Court of Appeals, and though the rule was subsequently revoked by the agency,[44] the court upheld EPA's broad assertion of authority under the section to obtain health and safety data on chemical substances, even during the research and development of a product and even though a company did not manufacture, process, or distribute a particular substance.[45] This broad conferral of power on EPA to collect information about a chemical even though it was not yet commercialized by a particular company may yield important health reasons why a company chooses not to pursue production, although another company may decide otherwise.

In September 1982, EPA reissued a rule implementing § 8(d).[46] The health and safety data reporting rule has two basic requirements. It requires the submission of unpublished health and safety studies on specifically listed chemicals by manufacturers, processors, and others in possession of them. This exempts distributors from reporting studies on designated substances, and it also relieves manufacturers and processors from submitting information contained in research and development and in underlying data such as medical records and exposure monitoring data on chemicals not on the TSCA chemical inventory. The 1982 rule required unpublished health and safety data to be submitted to EPA for asbestos and 39 chemicals recommended for additional testing by the ITC. In a related action, EPA also proposed a rule requiring data submissions on 14 chemicals recommended for testing by the ITC since June 1981.[47]

Commercial manufacturers and processors of a listed chemical (and those who are proposing to do so) are required to submit copies of both studies in their possession at the time the chemical is listed and lists of studies known to the submitter but not in his possession. (This does not require these parties, however, to update the studies.) Persons no longer manufacturing or processing a chemical when it is listed, but who manufactured or processed it or proposed to do so at any time during the time it was listed, must only submit copies of studies in their possession.

Substantial Risk Notices.—Under § 8(e) of TSCA, a company is required to notify EPA within 15 days of obtaining information that reasonably supports the conclusion that the substance or mixture presents a "substantial risk of injury to health or the environment. . . ." Very often these substantial risk notices concern occupational exposures and hence may be a very important source of data concerning chemicals associated with reproductive effects. Guidance on the submission of substantial risk notices was published by the Agency in September 1977.[48] In March 1978, EPA issued a policy statement interpreting the section.[49]

These notices are evaluated by EPA's Office of Pesticide Programs and Office of Toxic Substances. Referrals to other agencies, or decisions to list the chemical under a § 8 reporting rule to gather additional toxicity or exposure data or to undertake a formal risk assessment on the substance, follow. Section 8(e) submissions and initial evaluations are available for public inspection and copying. The agency thus far has published three volumes of initial evaluations covering approximately 500 notices received through December 31, 1982. A number of these contain preliminary information on reproductive health hazards. This information could be valuable in a product liability case brought by a worker exposed to a reported substance.

Section 10: Data Collection[50]

Section 10 requires the Administrator to conduct such research, development, and monitoring as is necessary to carry out the purposes of TSCA. Pursuant to this section, EPA has designed laboratory protocols and carried out some limited basic research on reproductive health hazards associated with chemicals. It also authorizes EPA to establish the Interagency Toxic Substances Data Committee (ITSDC), which is responsible for the development and coordination of a Federal chemical information system.[51] The goal of the ITSDC is systemized retrieval of toxicological and other scientific data that can be used for research, risk analysis, and decisionmaking under § 25(b). The Council on Environmental Quality (CEQ) and the Office of Toxic Integration are responsible for the day-to-day management of the Chemical Substances Information Network (CSIN).

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[43] 43 Fed. Reg. 30,984 (1978).

[44] 44 Fed. Reg. 77,470 (1979).

[45] Dow Chemical Co. v. EPA, 605 F.2d 673 (3rd Cir. 1979).

[46] 47 Fed. Reg. 38,780 (1982).

[47] 47 Fed. Reg. 38,800 (1982).

[48] 42 Fed. Reg. 45,362 (1977).

[49] 43 Fed. Reg. 1110 (1978).

[50] 15 U.S.C. §§ 2609, 2624 (1982).

[51] This committee assumed some of the data collection capabilities of the now defunct ILRG.

Technical Note #D.2: Cancellation of Pesticides Under FIFRA

Section 6(a): Automatic Cancellation[52]

FIFRA directs EPA to automatically cancel a pesticide registration 5 years after the registration date unless the registrant requests the continuance of the registration and EPA determines that the continued use of the product "will not have unreasonable effects on the environment." In order for EPA to make this determination, the registrant must submit data on the use, exposure, and health effects of the active ingredients in the pesticide, pursuant to 40 C.F.R. Part 158, and specific data requests by EPA (referred to as "call-ins"). The re-registration process, according to EPA officials, should eventually provide more health data on which to determine the health and environmental effects of pesticides that have been registered under FIFRA in prior decades. Under re-registration procedures initiated in 1984, EPA is specifically requesting teratology and multigenerational studies to determine reproductive effects.

Section 6(b): Cancellation Based on Findings of Unreasonable Adverse Effects[53]

EPA may initiate procedures to cancel a pesticide's registration or change its classification from general to restricted use if it appears that the pesticide, its labeling, or other material required to be submitted does not comply with the statute, or when used in accordance with widespread and commonly recognized practice, generally causes unreasonable adverse effects on the environment.[54] Various economic aspects are to be balanced by the Administrator against findings of adverse risk.[55] A decision to cancel must be made if reclassification of the pesticide to restricted use(s) will not adequately protect against those risks. The notice of the cancellation or reclassification must be mailed to the registrant and published in the *Federal Register* along with the regulatory impact analysis of the decision through the RPAR process. While this notice is generally geared to inform those who depend on the use of the particular pesticide of the Administrator's intent, it may also serve to alert the public to hazards associated with the substance. Unless the pesticide is designated as an imminent hazard (discussed below), the cancellation procedures may take several years to complete.

Section 6(d): Suspension[56]

FIFRA defines the term "imminent hazard" as "a situation that exists when the continued use of a pesticide during the time required for cancellation proceeding(s) would be likely to result in unreasonable adverse effects on the environment or will involve an unreasonable hazard to the survival of a species declared endangered or threatened by the Secretary pursuant to the Endangered Species Act of 1973."[57] Such unreasonable adverse effects on the environment, as discussed above, include hazards to human health.

On finding that action is necessary to prevent an imminent hazard during the time required for cancellation or change in classification proceedings, EPA may issue an order to suspend the registration of a pesticide immediately. (This recently happened when EPA suspended the registration of EDB due to groundwater contamination.) Concurrently, EPA must issue a notice of its intention to change the classification of a pesticide or cancel a registration. This notice must inform the registrant of the order and contain the Administrator's findings pertinent to the issue of imminent hazard.

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[52] 7 U.S.C. § 136d(a) (1982).
[53] 7 U.S.C. § 136d(b) (1982).
[54] Id.
[55] Id.
[56] 7 U.S.C. § 136d(c) (1982).
[57] 7 U.S.C. § 136d(b) (1982).

Appendix E

NRC Regulation of Exposure

Permissible Doses, Levels, and Concentrations

Section 20.101 of 10 C.F.R. establishes occupational radiation dose standards. These standards include only exposure received in the course of employment; a worker's exposure resulting from medical treatment or other non-job-related circumstances is not considered. Generally, a worker is not to receive a total occupational dose in excess of those in the following table:

Rems Per Calendar Quarter

Whole body; head and trunk; active blood-forming organs; lens of eye, or gonads	1.25
Hands and forearms; feet and ankles	18.75
Skin of whole body	7.50

Cumulative lifetime dosage is limited by the formula 5(N-18), where N is the worker's age at his or her last birthday. The purpose of this formula is to set an upper limit on cumulative lifetime dose, and it has a variable individual impact depending on the status of the employee. For a *permanent employee with a history of exposure*, application of the formula can result in an exposure limit of 1.25 rems per quarter; but for a *new regular employee* or *temporary employee, with no dose history*, the formula does not provide for a limiting level. In this latter case, a 3 rem quarterly standard would serve as the dose limit.[1]

Prior dose must be determined whenever an employee is likely to receive an occupational dose in excess of 25 percent of the standards specified in the above table.[2] This determination depends largely on the employee. The worker must sign a statement that he or she had no prior occupational dose during the current calendar quarter, or must describe the amount of any dose received during that quarter. Calculation of previously accumulated occupational doses is also required.

NRC limits exposure of workers who are under 18 years of age to a quarterly dose of 10 percent of the limits specified in the above table. Therefore, the maximum dose to the minor's whole body or gonads cannot exceed 0.125 rems per quarter.[3]

Worker exposure to concentrations of airborne radioactive materials and to radioactive materials capable of skin absorption is restricted. Internalization by either route in any calendar quarter cannot exceed material specific limits set forth in 10 C.F.R., Appendix B of Part 20. Appendix B contains additional restrictive limits for minors. The licensee is directed to ". . . use process or other engineering controls, to the extent practicable, to limit concentrations of radioactive materials in air. . . ."[4] When it is impracticable to use such controls, other precautionary procedures, including limitation of working time or provision of respiratory protective equipment, is mandated.[5] Inhalation protection is important, since many radioactive materials used in the nuclear industry cannot damage reproductive systems unless internalized.

Precautionary Procedures

There are three main precautionary procedures required of all NRC licensees that influence reproductive health. First, licensees must make periodic surveys to evaluate the extent of radiation hazards. Surveys are an evaluation of the hazards incident to the presence of radioactive materials, and include measurements of radiation levels.[6]

Second, the licensee must supply employees with monitoring equipment and must require its use. Workers who must wear monitors are differentiated by age. Any adult likely to receive 25 percent, and any minor likely to receive 5 percent, of the values specified in the above table must wear monitors.

Third, the licensee is required to clearly mark restricted areas, which must be controlled for the purpose of protecting workers from exposure. The design of warning devices is prescribed, and radioactive containers and access points to radiation areas must be clearly marked.[7]

Records, Reports, and Notification

All licensees are required to maintain records of radiation surveys and personnel exposure. Yearly reports to the NRC are required only for licensees in the industries thought to encompass the greatest exposure. These industries include nuclear reactors, radiography, fuel processing, high-level waste repositories, spent fuel storage, and facilities using specified quantities of byproduct material.[8] Reports must detail the number of workers monitored and provide a statistical summary of their exposures. This group of licensees must also report to the Commission on the exposure of each worker, at termination of employment.[9] All licensees must also report to NRC any condition that results in over-exposure of any workers.[10]

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[1] 10 C.F.R. pt. 20, § 20.101(b)(1) (1984).

[2] 10 C.F.R. pt. 20, § 20.102.

[3] 10 C.F.R. pt. 20, § 20.104.

[4] 10 C.F.R. pt. 20, § 20.103(b)(1).

[5] 10 C.F.R. pt. 20, § 20.103(b)(2).

[6] 10 C.F.R. pt. 20, § 20.201

[7] 10 C.F.R. pt. 20, § 20.203.

[8] 10 C.F.R. pt. 20, § 20.408.

NRC regulations also provide for reports to workers and for NRC inspections of facilities. Required reports to workers include both general instructions and individual exposure data.

The licensee is required to make general information available to workers, including copies of the license, license conditions, licensed operating procedures, and notices of violations involving radiological working conditions.[11] Furthermore, the licensee must instruct employees in the health protection problems associated with exposure to radioactive materials.[12]

Licensees must furnish a written report to workers describing their personal radiation exposure data, including any analysis of radioactive material retained by the body. These reports are to be made annually or on termination of the worker, but only at the worker's request. A former employee can request an exposure report from the employer's records.[13]

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[9] 10 C.F.R. pt. 20, § 20.408(b).

[10] 10 C.F.R. pt. 20, § 20.403.

[11] 10 C.F.R. pt. 19, § 19.11.

[12] 10 C.F.R. pt. 19, § 19.12.

[13] 10 C.F.R. pt. 19, § 19.13.

Appendix F

List of Contractor Reports, Working Papers, and Staff Papers

The following is a list of contractor reports and staff papers that are available from:

National Technical Information Service (NTIS)
U.S. Department of Commerce
Springfield, VA 22161

Office of Technology Assessment (OTA)
United States Congress
Biological Applications Program
Washington, DC 20510

Michael S. Baram:
"Regulation of Ionizing Radiation by the Nuclear Regulatory Commission"

Brenda Eskenazi:
"Reproductive Hazards of Chemical Exposures in the Workplace"
"Chemical Hazards to Human Reproduction in the Workplace"

Environmental Law Institute:
"EPA Authority and Activities Relating to Occupational Reproductive Hazards"

Erwin Goldberg:
"Tests of Male Reproductive Function"

E. Marshall Johnson:
"Mechanisms of Action, Assays Of, Defined and Dose Related Effects Of, Agents Affecting Developmental and Reproductive Parameters in Humans and Animals"

Michael Rosenberg:
"Epidemiologic Surveillance of Occupational Effects of Reproduction"

Mark A. Rothstein:
"The Regulation of Reproductive Hazards Under OSHA"

Joseph Santodonato:
"Workplace Physical Factors Affecting Human Reproductive Function"
"Effect of Workplace Chemicals on Reproduction Function in Laboratory Animals"

Staff paper (available from OTA only):
"Selected Aspects of Reproductive Health Hazards Regulation"

The following contractor reports have been prepared as working papers, available from NTIS and OTA (Biological Applications Program).

- *Selected Ethical Issues in the Management of Reproductive Health Hazards in the Workplace which contains:*

—Ronald Bayer:
"Policy Options Before Congress"
"Ethical Issues in Risk Assessment and the 'Right to Know' "
"The Moral Issues"
—Tom Beauchamp:
"Ethical Issues in Discrimination and Job Termination"
—James Childress:
"Ethical Analysis of Legislative Option: Compensation for Reproductive Damage Resulting From the Workplace"
—Thomas Murray:
"Ethics, Risk Assessment, and Reproductive Hazards"

- *Reproductive Hazards in the Workplace: Foreign Laws and International Agreements,* by Michael S. Baram

Appendix G

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Index

Index

acrylamide, 255
acrylonitrile, 88, 89, 90, 197
adriamycin, 269
AFL-CIO, 193, 390
AFL-CIO v. *Brennan*, 396
AFL-CIO v. *Marshall*, 397
Agent Orange (2,4,5-tricholorphenoxyacetic), 7, 77, 78
agricultural chemicals, 74-75, 110
  exposure to, 74, 92
Alabama, 245
Alaska, 206
alcohol consumption, 2, 32, 57, 60, 61, 81, 101, 162, 165, 167, 170, 345, 347, 350, 356, 357, 365
allyl chloride, 83
alpha-fetoprotein (AFP), 147, 154, 155
American College of Obstetricians and Gynecologists, 106
American Conference of Governmental Industrial Hygienists, 100, 204, 254, 269
American Cyanamid Co., 184, 187, 191, 251-260
  Occupational Exposure Review Committee (OERC), 256, 258, 260
American Federation of Labor, 236
American Federation of State, County, and Municipal Employees, 201, 390
American Iron and Steel Institute, 193
American Iron and Steel Institute v. *OSHA*, 396
American Law Institute, 304
American Medical Association, 106
American National Standards Institute, 189
American National Standards Institute Committee C95, 98
American Petroleum Institute, 164, 193, 394
American Petroleum Institute v. *OSHA*, 391
American Textile Manufacturers Institute v. *Donovan*, 397
Americium, 224
aminopterin, 170
anesthetic agents, 7, 82, 83, 110, 170
animal studies, 8, 54, 67, 70-91, 95, 96, 98-104, 108, 110, 162, 167-71, 175, 202, 208, 217, 218, 252, 253, 255, 288, 344, 346, 355, 356, 395
antihistamines, 356
antimony, 7, 69, 74, 110, 204
  uses of, 74
Apgar score, 45, 106
Aristotle, 33
aromatic amines, 88
arsenic, 7, 69, 73, 74, 93, 110, 169, 170, 390, 392, 395
  occupational exposure, 73
  occurs in, 73
asbestos, 192, 196, 197, 292, 305, 306, 402, 403
  standard 185, 197, 391
  emergency temporary standard, 395
Asbestos Information Association v. *OSHA*, 196
asbestosis, 25, 281-286, 291, 305, 306
"as-low-as-reasonably achievable" (ALARA) assumption, 226, 228, 230
Assistant Secretary for Health, DHHS, 183
Atlanta, Georgia, 184
atmospheric pressure, 93, 94, 102, 103, 110
Atomic Energy Commission (AEC), 223, 224
  Standards for Protection Against Radiation, 223

Belmont Report, 332
Bendectin®, 356
benzene, 7, 10, 81, 82, 169, 203, 269, 394
  standard, 193, 197
Benzene, 196, 393-395, 397
beryllium, 69
biological agents, 107-110
birth defects (see congenital malformations)
Birth Defects Monitoring Program (BDMP) (see Centers for Disease Control (CDC))
birth rate, 34
bisulfan, 170
Bladex (see cyanazine)
boron, 7, 69, 70, 71, 110
  as boric acid, borates, 70
  uses of, 70
Myra Bradwell, 235
Bradwell v. *Illinois*, 236
breast milk, 53, 74, 77, 79, 80, 151, 293, 354, see also lactation
Bulletin of Atomic Scientists, 229
Bureau of Labor Statistics (BLS) (see Department of Labor)
butadiene (1-3 butadiene), 7, 67, 88, 89, 110, 175, 210, 222
byssinosis, 286, 394, 395

cadmium, 7, 69, 72, 73, 93, 110, 203, 269
  occupational exposure to, 72
  uses of, 72
caffeine, 357
California, 76, 163, 186, 206, 236, 239, 316, 317
Canada, 252
capafol (Difolatan®), 269
captan, 269
carbaryl, 7, 67, 75, 110, 269
  workers exposed to, 75

carbon dioxide, 97
carbon disulfide, 7, 67, 81, 82, 93, 169, 175, 204
carbon monoxide, 169, 269
carbon tetrachloride, 7, 81, 82, 269, 309
Carter Administration, 10, 183, 195, 204, 209, 392, 393, 395, 396
Centers for Disease Control (CDC), 22, 27, 90, 164, 172, 174, 182, 183, 184, 208, 353
  Birth Defects Monitoring Program (BDMP), 27, 90, 164, 351, 352
central nervous system (CNS), 46, 55, 57, 70, 72, 90, 147, 154, 170, 352, 353, 356, 357
Chemical Information System (NIH), 171
Chemical Manufacturers Association, 193
chemicals
  quantities manufactured, 37
  chemical work, 92
Chemical Substances Information Network (CSIN), (EPA, CEQ), 171, 403
chemotherapeutic drugs, 175
chlamydia, 344
chlorambucil, 269
chloramphenicol, 356
chlordane, 92
chlordecone, 110
chlorinated terphenyls, 402
chlorohenothane, 77
chloroprene, 7, 67, 88, 89, 110, 169
chromium, 69
Cleveland Board of Education v. *LaFleur*, 240, 241
Clyne, Dr. Robert, 259, 260
cobalt, 73, 93
cold environments, 94, 110
congenital malformations, 57, 60-62, 72, 73, 77, 71, 82, 89, 90, 92, 93, 96, 100, 106, 107, 131, 147, 148, 150, 152, 155, 165, 167, 170, 174, 198, 238, 239, 302, 310, 343, 348-357, 400
  incidence of, 5, 33
  terminology, 55
congenital syphilis, 109, 111
congressional issues and options:
  compensation for job-induced reproductive harm, 24-26
  issues in research, 26, 27
  regulation, 21-24
  sex discrimination, 16-21
Connecticut, 206, 207
Consumer Product Safety Commission (CPSC), 172, 173, 204, 216, 219, 398
control of hazardous exposure:
  administrative controls, 4, 198, 267
  educational programs, 4
  engineering controls, 4, 32, 198, 248, 260, 267
  personal protective equipment, 32, 198, 248, 260, 263, 267
copper, 69, 73
Costle, Doug, 200
cotton dust standard, 185, 186, 187, 391, 397
Council on Environmental Quality (CEQ), 171, 403
Craig v. *Boron*, 240
cyanazine (Bladex), 217, 218
cyanide gas, 304
cyclophosphamide, 269
cyclopropane, 82, 83
cystic fibrosis, 347
cytomegalovirus, 7, 108, 110, 355

dacarbazine, 269
danazol, 356
data:
  Chemical Information System, NIH, 171
  Environmental Mutagen and Environmental Teratogen Information Center, DOE, 172
  Chemical Substances Information Network (CSIN), EPA, CEQ, 171, 403
  Interagency Toxic Substances Data Committee (ITSDC), EPA, 403
  Scientific Parameters in Health and the Environment, NIH, 171
  Status Report of Chemical Activities, EPA, 173, 219
  Toxic Information Series, EPA, 173
DBCP (dibromochloropropane), 4, 6, 7, 36, 48, 74, 75, 76, 110, 163, 169, 175, 197, 199, 200, 204, 208, 217, 295, 296
  uses of, 75
DDT, 7, 53, 74, 76, 77, 110, 344, 346, 355
  use of, 76
Delaware, 206, 351
Department of Agriculture, 173
Department of Defense (DOD), 94, 225, 270, 272
Department of Energy (DOE), 94, 171, 172, 225
  Environmental Mutagen and Environmental Teratogen Information Center, 172
Department of Health and Human Services (DHHS), 56, 172, 174, 182, 195, 225
Department of Labor, 19, 22, 174, 181, 184, 195, 197, 392
  Bureau of Labor Statistics (BLS), 33, 35, 185
  Office of Federal Contract Compliance Programs (OFCCP), 19, 205
  Standard Industrial Classification (SIC) injury and illness rates, 184
Department of Transportation (DOT), 225
Department of the Treasury
  Bureau of Alcohol, Tobacco, and Firearms (BATF), 398
DES (see diethylstilbestrol)
developmental toxicants/toxicity, 5, 6, 55-57, 59, 62, 74, 161, 169, 170, 217, 218, 219, 227, 228

  dose-response considerations
  principal manifestations, 61
  species/gender differences, 59
  time frame, 59
diabetes, 347, 355
diamox, 256, 257
diazinon, 269
dibromochloropropane (see DBCP)
dibutylphthalate, 269
dichloropropene (1,3-dichloropropene), 83
dicumarol, 356
Dieldrin, 77
diethylstilbestrol (DES), 32, 91, 269, 320, 356
dimethylacetamide (DMAC), 265, 269
diethylformamide (DMF), 265
dimethylphthalate, 269
dimitrotoluene, 67
dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin) (TCDD), 7, 77, 78, 81, 110, 175, 269, 354
  as a contaminant, 77
diphenylamine, 269
diphenylhydantoin, 170
diquat, 269
discriminatory impact (disparate impact), 242-247, 249-251, 262, 263
  business necessity exception, 243-250
  statistical analysis to prove, 262-263 (applicant flow data, population pool analysis)
discriminatory treatment (facial and pretext discrimination), 242-251
  bona fide occupational qualification (BFOQ), 242-246, 250, 262
District of Columbia, 206, 351
DNA (deoxyribonucleic acid), 56, 72, 86, 109, 110, 154
dose-response assessment, 8, 37, 162, 170, 395
Down syndrome, 60, 147, 154, 167, 352, 355, 357
Dun & Bradstreet, 175, 176
Dunlop, John (former Secretary of Labor), 193

EDB (ethylene dibromide), 7, 84, 85, 110, 169, 175, 204, 404
  exposure to, 84
  uses of, 84
Egypt, 395
E.I. du Pont de Nemours & Co., 255, 265
electric fields, 93, 94
Eli Lilly & Co., 217
embryo/fetus, 5, 14, 15, 49, 51, 52, 54-56, 57, 61
  abnormal development, 54
  development, 51, 52
  duty to, 15
  embryogenesis, 49
    impaired, 56
  embryotoxicity, 55, 57
  fetotoxicity, 55, 73, 74
  loss of embryo, 5, 51, 54, 61 (see also spontaneous abortion)
  rights of, 14, 15
emergency temporary standards (ETSs), 9, 23, 24, 182, 194, 195, 196, 197, 199, 208, 209
  "grave danger," 23, 182, 194, 196, 197, 208
employer and employee duties, 187-189
  biological monitoring, 188
  compliance with standards, 187
  environmental monitoring, 188
  medical surveillance, 188
Endrin, 77
enflurame, 82, 83
England, 102
Environmental Mutagen and Environmental Teratogen Information Center (database), (DOE), 172
Environmental Protection Agency (EPA), 9-11, 21, 27, 35, 37, 38, 55, 56, 75, 77, 163, 171-173, 176, 181, 182, 184, 200-204, 209-225, 398, 400-404
  Advanced Notice of Proposed Rulemaking (ANPR), 218
  Cancer Assessment Group, 374
  Developmental Biology Division in Research, 172
  Federal Radiation Guidance, 223, 225
  Health Effects Research Laboratory (HERL), 172
  Interagency Toxic Substances Data Committee (ITSDC), 403
  Male/Female Reproductive Effects Risk Assessment Guidelines, 9, 173
  Office of Pesticide Programs (OPP), 172, 173, 215, 222, 403
  Office of Radiation Programs, 172, 173
  Office of Research and Development (ORD), 172
  Office of Toxic Integration, 403
  Office of Toxic Substances (OTS), 171-173, 218, 403
  Pesticide Advisory Committee, 200
  Proposed Guidelines for Assessment of Developmental Toxicants, 9
  Rebuttable Presumption Against Registration (RPAR), 201, 202, 215, 404
  red-border review of regulatory actions, 172
  Reproductive Effects Assessment Group (REAG), 173, 218, 221
  Toxics Information Series, 173
epichlorohydrin, 7, 67, 83, 84, 88, 89, 92, 110
epidemiological studies, 8, 68, 163-167, 205, 208, 245, 292, 395, 402
  analytical, 164
    case control, 164

    - cohort, 164
    - cross sectional, 164
  - descriptive, 164
    - case reports, 164
    - surveillance, 164
  - general considerations, 165
    - measurement of reproductive endpoints, 165
    - study design, 165
  - key factors, 166
    - confounding factors, 166
    - power, 166
    - sample size, 166
  - major factors in discrepancies among, 68
- Equal Employment Opportunity Commission (EEOC), 19, 184, 205, 262, 263
- equal protection analysis, 237-246
  - rational basis test, 238-240
  - strict scrutiny test, 238-240
- ergotamines, 269
- ethanol, 81
- ethical issues, 4, 14, 15, 329-337
  - beneficence, 14, 15, 329, 331-335, 337
  - employer's duty to workers, 15
  - justice, 4, 14, 15, 329, 331, 335, 336
  - respect for persons, 14, 329, 331, 332, 334, 335, 337
- ethinyl estradiol, 90, 91
- ethoxy ethanol, 265
- ethyl benzene, 69
- ethylene dibromide, 269
- ethylene dichloride, 92
- ethylene oxide (EtO), 4, 6, 7, 10, 36, 67, 85-87, 175, 198, 199, 201-203, 217, 219, 222, 269, 390, 392
  - occupational exposure to, 85
  - standard, 198, 203, 208
  - uses of, 85
- ethylene thiourea (ETU), 7, 67, 88, 89, 110, 204, 265, 269
- EtO (see ethylene oxide)
- exposure assessment, 8, 37, 162
  - estimates of exposed workers, 162
- Exxon Chemical Americas, 265, 266, 267

- Federal Advisory Council on Occupational Safety and Health (FACOSH), 195
- Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) (see legislation)
- Federal Radiation Council (FRC), 223, 225
- female reproductive health, tests of, 132, 139-151, 154, 343, 346
  - basal body temperature, 142, 154
    - patterns of, 144
  - cervical mucus, 139, 142, 145, 154, 343, 346
    - sperm-cervical mucus interaction, 139
  - diagnostic techniques, 134
  - endometrial cells, 145
    - endometrial biopsy, 142, 145, 146
  - indirect indicators, 142, 144
  - menstrual cycle, 143 (see also menstruation)
  - ovaries (see ovarian function)
  - patient history, 132
  - physical examination, 141
  - tubal patency/uterine structure, 146
- fertility evaluation, 129-151, 365
  - features of, 129
  - chronology of, 130
  - patient history questionnaire, 365
    - (see male reproductive health, tests of, and female reproductive health, tests of)
- fetal:
  - growth, 49, 51, 52, 57
    - impaired, 56
  - loss, 54
  - measurements
    - amniocentesis, 147, 148, 155
      - karyotyping of amniotic cells, 154
    - chorionic villus biopsy, 148, 149
    - contraction stress test, 155
    - fetoscopy, 148, 155
    - heart response, 155
- fetal protection policies (FPPs), 4, 12, 16-19, 21, 245-247, 249, 250, 251-261, 267, 331, 335, 337
- fetal solvent syndrome, 82
- Finland, 68, 73, 93
- 5, azacytidine, 269
- Florida, 206, 294, 351
- fluorinated hydrocarbons, 82, 83
- Food and Drug Administration (FDA), 27, 172, 173, 204, 216, 398
  - Center for Devices and Radiological Health, 100
- Ford Administration, 204, 392, 393
- formaldehyde, 7, 10, 87, 88, 110, 204, 210, 222
- formamide, 265
- Fortune 500, 235, 261
- 4',4' methyene dianiline, 222
- 14th Amendment to the U.S. Constitution, 235, 237
- France, 164

- *GAF Corp. v. OSHRC*, 390
- gamma rays, 94, 355
- *Geduldig v. Aeillo*, 241
- General Accounting Office, 174, 401
- Georgia, 206, 311, 315, 351
- glial protein S-100, 154
- glycidyl ethers, 67
- glycol ethers, 10, 67, 175, 210, 218, 219, 222, 355
- gonorrhea, 344, 346
- gravitational fields, 93, 94
- Greece, 395

halothane, 82, 83
Hamilton, Alice, 35
Hawaii, 75, 76, 108, 200
Hayes v. *Shelby Memorial Hospital,* 245, 246, 247, 248, 249
hazard identification, 8, 37, 161
Health Effects Research Laboratory (HERL), EPA, 172
heat, hot environments, 69, 94, 103, 110
hemophilia, 147, 148, 154, 155
hepatitis B, 7, 108, 109, 111, 355
Heptachlor, 77
heroin, 347
herpes simplex virus, 109, 111, 355
herpes zoster, 355
hexafluoroacetone (HFA), 265, 269
"hierarchy of controls" concept, 198
Hiroshima, 96
hormones, 7, 43-49, 53, 58, 76, 77, 79, 90, 91, 110, 131, 142, 148, 152-155, 174, 175, 199, 295, 343, 345-347, 356
  follicle-stimulating hormone (FSH), 44-48, 152
  luteinizing hormone (LH), 44-48, 152, 199
  luteinizing hormone-releasing hormone (LHRH), 44-47
  occupational exposure to, 90, 91
human chorionic gonadotropin (hCG), 45, 49, 50, 51, 146, 154
hydrazine hydrate, 256
hydrazine sulfate, 256
hydrocortisone acetate, 91
hydrogen sulfide, 69, 70, 93
hyperbaric/hypobaric environments, 102, 103, 110, 197, 391

Illinois, 206
imipramine, 356
indium, 269
industrial alcohols, 175
industrial exposures, undefined, 91, 110
Industrial Union Department v. *American Petroleum Institute (API),* 393
infant mortality, 60
  rates of, 348, 349, 350, 351
infertility, 4, 31, 33, 59-62, 70-72, 75, 76, 83, 93, 96, 129, 152, 163, 165, 252, 259, 289, 293, 296, 297, 301-348, 402
infrared radiation, 97
insulin, 356
interagency relations, 183
Interagency Testing Committee, 400, 403
International Chemical Hazards Union, 217
International Chemical Workers Union, 275, 276
International Commission on Radiation Protection (ICRP), 226, 227, 230

ionizing radiation (see radiation)
Iowa, 206, 351
isoretinoin (Accutane®), 356
Israel, 76, 92
ITT v. *Grinnell,* 391

Judges 13:7, 33

Kansas, 351
Kentucky, 294
Kepone (chlordecone), 7, 74, 77, 169
  use of, 77
Klinefelter syndrome, 147, 154, 347

laboratory work, 7, 92, 175
lactation, 53, 141 (see also breast milk)
laparoscopy, 144, 146
  for observation of ovary, 144
  for observation of peritoneal cavity, 146
  laparoscopic ovarian biopsy, 144
laser radiation, 7, 97, 98
lead, 4, 6, 7, 35, 36, 69, 70, 72, 110, 169, 170, 175, 184, 186, 188, 198, 199, 253, 256, 257, 258, 260, 265, 269, 351, 355, 390, 392
  lead compounds, 69
  lead standard, 198, 200, 203, 208, 391, 394, 397
  workers exposed to, 69
lead chloride, 70
legislation:
  Atomic Energy Act (AEA), 171, 209, 223, 224, 225
  Civil Rights Act (see Title VII of the Civil Rights Act)
  Clean Air Act, 171, 202, 210, 224
  Comprehensive Environmental Response, Compensation and Liability Act of 1980 (Superfund), 171, 210
  Endangered Species Act of 1973, 404
  Energy Reorganization Act of 1974, 223, 224
  Environmental Research and Development Act, 172
  Federal Employee Compensation Act, 272
  Federal Environmental Pesticide Control Act (FEPCA), 184
  Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), 3, 10, 171, 172, 182, 184, 200, 201, 209, 210, 231-218, 219, 221, 222, 404
    cancellation and reregistration of pesticides, 216
    farmworker protection standards, 215
    registration, 214
    registration of pesticides, 214
    regulatory action on applications for registration, 214
    special review, 214

    storage, packaging, and disposal of pesticides, 216
    use of restricted pesticides, 216
  Federal Mine Safety and Health Act, 174
  Federal Tort Claims Act, 231, 325, 326
  Federal Water Pollution Control Act of 1972, 210
  Mine Safety and Health Act, 219, 398
  National Environmental Policy Act, 193
  National Labor Relations Act, 184
  Occupational Safety and Health Act (OSH Act), 3, 4, 9, 10, 15, 21, 22, 24, 38, 174, 181-209, 219, 332, 334, 394
    action level concept, 188
    general duty clause, 189-192
    hazard communication standard, 206
    hazard exposure control, 198-199
    hazard identification, 195-197
    Written Hazard Communication Program, 207
  Paperwork Reduction Act, 220
  Pregnancy Discrimination Act, 242, 244, 248
  Regulatory Flexibility Act, 193, 220
  Resource Conservation and Recovery Act, 171, 210
  Safe Drinking Water Act, 210
  Solid Waste Disposal Act, 210
  Title VII of the Civil Rights Act, 3, 4, 12, 15, 16, 18, 21, 38, 184, 186, 187, 295, 239, 241, 242, 244, 245, 246, 250, 251, 260, 261, 263
  Toxic Substances Control Act (TSCA), 3, 6, 8, 10, 37, 38, 56, 62, 171, 172, 210-213, 216-218, 219, 221, 222, 267, 400-403
  Uranium Mill Tailings Radiation Control Act of 1978, 224
lithium, 69, 356
Louisiana, 206, 351

magnetic fields, 93, 94, 101, 102
Maine, 206
male reproductive health, tests of, 46, 47, 131-140, 152, 153
  antisperm antibodies, 152
  automated analysis of sperm movement, 153
  diagnostic techniques, 133
  immunobeads, 152, 153
  laboratory evaluation, 135
  Leydig cells, 46, 47
  personal history, 131, 132
  physical examination, 131
  semen (see semen)
  sperm (see sperm)
  testicular biopsy, 152
  vasography, 152
manganese, 7, 69, 71, 110
  compounds of, 71
  uses of, 71
Manville Corp., 321, 322
marijuana use, 345, 346, 347, 357
Maryland, 206, 351
Massachusetts, 206, 207
McCarthy Scales of Children's Abilities, 80
medical removal protection/policy (MRP), 185, 186, 198, 199, 200, 390
  rate retention (RR), 185, 186
melphalan, 269
Memorandum of Understanding, EPA, OSHA, 219, 221, 222
Memorial Sloan-Kettering Cancer Center, 268
menstruation, 142, 143, 153
  anovulatory cycle, 153
  menstrual cycle, 143
  regular menstruation, 142
Mercer, Glen, 257
mercury, 6, 7, 57, 69, 71-73, 110, 203, 269, 355
  forms of, 71
  methylmercury, 32, 69, 170
  occupational exposures, 71
Merrell-National Laboratories, 356
metals:
  biological indicators of exposure, 69
  classified as occupational carcinogens, 69
methotrexate, 170, 256, 269
methoxychlor, 344
methoxyflurane, 269
methylcellosolve, 269
methyl chloride, 92
Metropolitan Atlanta Congenital Defects Surveillance Program, 164, 352
Michigan, 79, 206, 295, 311
microwave radiation (see radiofrequency/microwave radiation)
Minamata disease, 32, 355
Mine Safety and Health Administration, 181, 204
Minnesota, 206, 294, 351
Mirex, 77
Mississippi, 351
Missouri, 206, 351
monohalomethanes, 67
Mount Sinai School of Medicine, 101
Muller v. Oregon, 237
mumps, 6, 152, 346, 347, 355
mutagens/mutagenicity, 56, 77, 81, 83, 84, 86, 90, 202, 218, 219, 252, 255, 275, 296, 302, 322, 330, 400, 401

Nagasaki, 96
narcotics use, 345, 346
National Academy of Sciences, 37, 172
National Advisory Committee on Occupational Safety and Health (NACOSH), 195

National Center for Health Statistics (NCHS), 348, 352
National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research, 332
National Congress of Hispanic American Citizens v. *Marshall*, 392
National Council on Compensation Insurance (NCCI), 305, 306
National Council on Radiation Protection and Measurements (NCRP), 226, 227, 228, 230, 247
National Fire Protection Association, 189
National Institute for Environmental Health Sciences (NIEHS), 27
National Institute for Occupational Safety and Health (NIOSH), 5, 9, 10, 22, 23, 27, 67, 69, 87-89, 101, 162, 163, 171, 174-176, 181-184, 189, 195, 198, 199, 204, 205, 208, 209, 255, 260, 276, 392, 393, 396, 400
  exposure estimates, 175
  Health Hazard Evaluation Program,174
  major activities, 174
  reproductive health hazard research 174, 175
  reproductive risk assessment, 175
National Institutes of Health (NIH), 27, 171, 173, 199
National Labor Relations Board, 184
National Occupational Hazard Survey 1972-74, 162
National Realty v. *OSHRC*
National Science Foundation (NSF), 27
National Toxicology Program (NTP), EPA, PHS, DHHS, 172, 175
NCHS National Health and Nutrition Examination Survey (HANES), 27
Nebraska, 351
New Hampshire, 206
New Jersey, 206, 207, 308, 399
New York, 206, 217, 237, 239
nickel, 69
9 to 5 Association of Working Women, 101
nitrophen (TOK), 218
nitrous oxide, 82, 83, 204, 269
noise, 94, 104, 110
nonionizing radiation, 96-99, 110
No-observed-effects-level (NOEL), 17, 170
  thresholds for toxic effects, 170
North Carolina, 206, 291, 351
North Carolina Workers' Compensation Act, 291
North Dakota, 351
nuclear magnetic resonance, 150
Nuclear Regulatory Commission (NRC), 9, 11, 21, 38, 94, 181, 209, 223-231, 269, 406
  regulations and reproductive risk, 226
  Regulatory Guide Number 8.13, 228, 230
  temporary workers, 308

Oak Ridge National Laboratory, 171
Occidental Chemical Co., 199
Occupational Safety and Health Act (OSH Act) (see legislation)
Occupational Safety and Health Administration (OSHA), 9, 10, 20-24, 35, 38, 69, 171-176, 181-189, 191-198, 200-204, 206-209, 215-217, 219, 221-223, 249, 251, 255, 257, 260, 271, 273, 274, 276, 279, 318, 390-399, 400
  Directorate of Technical Support, 183
  Office of Standards Review, 393
Occupational Safety and Health Review Commission (OSHRC) 10, 181, 182, 189, 191, 192, 209, 257, 391
Office of Management and Budget (OMB), 183, 195, 202, 219, 220
Ohio, 317
Oil, Chemical, and Atomic Workers Union, 193
oil industry workers, 92
1-3 butadiene, 7, 67, 88, 89, 110, 175, 210, 222
1,3-dichloropropene, 83
1,1,1,-trichloroethane (see DDT)
"Operation Ranch Hand," 78
optical radiation, 94
Oregon, 206, 237
organic compounds, 175
organic solvents, 81, 82, 89, 110
Organized Migrants in Community Action v. *Brennan*, 222
organophosphorous pesticides, 182, 197
organo-tin compounds, 175
oryzalin, 175, 217
osteoporosis, 31
OTA, 101, 258, 260, 263, 401
ovarian function, 141-144
  direct indicators, 144
    laparoscopy
    laparoscopic ovarian biopsy
    ultrasonography
  indirect indicators, 142-144

paraquat, 269
parathion, 269
PBB (polybrominated biphenyls), 7, 58, 78-81, 110, 402
PCB (polychlorinated biphenyls), 7, 53, 58, 67, 78-81, 110, 169, 170, 175, 204, 354
pelvic inflammatory disease (PID), 344, 345
penicillin, 356
Pennsylvania, 90, 206, 309
Pennsylvania Supreme Court, 20
perchlorethylene, 92
Permissible Exposure Limits (PELs), 188, 255
Peru, 102

pesticides (see chemical name)
  reregistration of, 171
Phelps Dodge Corp. v. *OSHRC*
phosphate, 402
physical agents, 93-104
polio, 355
polycyclic aromatic hydrocarbons, 344, 355
polyvinyl chloride (PVC), 89
Porter v. *Lassiter*, 311
Pott, Percivall, 35
pregnancy, 51, 73, 80, 81, 106, 107, 130, 140, 146, 165, 240, 241
  conception rate, 51
  delivery and lactation, 150
  discrimination on the basis of, 240, 241
  fertilization, 49, 51
  implantation, 50, 51, 73, 80, 81, 130, 140, 146, 165
  increase in blood volume, 51
  pregnant workers, 106, 107
  the pregnant woman, 49-51
Premanufacture Notification (PMN), 37, 163, 171, 211, 401
President Jimmy Carter, 220
President Gerald R. Ford, 220
President Ronald Reagan, 220
procarbazine, 269
product liability (see tort liability)
Proposed Guidelines for Assessment of Developmental Toxicants, EPA, 9
Proposed Interpretive Guidelines on Employment Discrimination and Reproductive Health Hazards, 205
puberty, 53, 54
Public Citizen Health Research Group, 201, 202
Public Health Service, 172, 182, 208
pulp and paper work, 92, 93
PVC (see polyvinyl chloride)

Quality of the Workplace Study (1977), 36

radiation, 4, 6, 7, 11, 31, 46, 93-96, 100, 167, 203, 223-231, 247, 269, 281-287, 345, 347, 355, 356, 405
  occupational exposure, 223
  temporary workers, 11, 228-230, 308, 405, 406
radiofrequency/microwave radiation, 7, 94, 98, 99, 175, 272
Rand Corp., 305
Reagan Administration, 10, 183, 209, 392, 395
Rebuttable Presumption Against Registration (RPAR), EPA, 201, 202, 215, 404
recombinant DNA, 7
Reed v. *Reed*, 239
reproductive function:
  aspects of, 43, 61
  assessment (measurement) of, 5, 44, 45, 61
  (see also female reproductive function)
  (see also male reproductive function)
reproductive endpoints, 44, 165, 167, 290, 365
  measurement of, 165
  population estimates of, 165
reproductive health protection policies, 263-276
reproductive toxicants/toxicity, 5, 6, 57-59, 68, 161, 169, 219
  classifications, 68
  gender/species differences, 59
  mechanisms of action, 68
  selected examples, 169
  time frame, 59
Research Triangle Park, NC, 172
Rhode Island, 206, 311
"right-to-know" laws, 206, 207, 396
risk:
  assessment, 7, 8, 31, 37, 161, 393, 395
    data use in, 163, 164
    in government agencies, 171
  characterization, 8, 38, 162
  management, 7, 8, 38, 161, 395
  worker perception of, 36
Risk Management Council, 221
Roche Laboratories, 356
Rockville, Maryland, 184
Roe v. *Wade*, 241, 311, 312, 313, 314, 315, 326, 331
rubber, 88, 89, 110
Rubber Manufacturers Association, 193
rubella, 6, 7, 107, 108, 111, 163, 355
Ruckelshaus, Willliam, 221
Russia, 74, 88, 89

Scientific Parameters in Health and the Environment (database), NIH, 171
scrotal cancer, 35
Secretary of Health and Human Services, 183
Secretary of Labor, 181, 183, 190, 191, 192, 194, 195, 393, 394, 396, 400, 404
selenium, 69
semen, 135-136, 175, 346 (see also sperm)
  ejaculate, 136
  laboratory evaluation, 135
  quality, 135
Senate Environment and Public Works Committee, 219
Service Employees International Union, 101
Seveso, Italy, 32
sex discrimination, 11, 16-21, 235-276 (see also

discriminatory treatment, discriminatory impact)
Shell Oil Co., 264, 265
sickle-cell anemia, 147, 154, 344, 347, 355
silicosis, 281-287, 291
Silvex (2-[2,4,5-trichlorophenoxy] proprionic acid), 77
smoking, 31, 32, 61, 73, 87, 94, 101, 129, 162, 167, 197, 345, 350, 351, 357, 365
Society of the Plastics Industry, Inc. v. *OSHA*, 396
South Carolina, 351
South Dakota, 351
Soviet Union, 68
sperm, 136-140, 153, 343, 346-348, 357 (see also semen)
  cervical mucus penetration, 139
  characteristics, 136-138
  function, 138
  post-coital test, 153
  single-image photomicrography/high speed cinemicrography, 153
  sperm-oocyte interaction, 139
  videomicrography, 153
  zona-free hamster egg penetration test, 140, 153
spontaneous abortion, 51, 53, 56, 61, 70, 72, 74, 81, 83, 86, 89, 93, 100, 149, 165, 167, 169, 198, 202, 297, 342-345, 346, 356
Standard Industrial Classification (SIC), (BLS, DOL)
  injury and illness rates, 184
Status Report of Chemical Activities data retrieval system, (EPA), 173, 219
sterility (see infertility)
streptomycin, 356
stress, 31, 105, 106, 110, 293
styrene, 7, 81, 82, 88-90, 169
Sudan, 395
sudden infant death syndrome, 350
sulfur, 73
sulfur dioxide, 69, 73, 93
Superfund, 171
Supreme Court, 185, 186, 188, 196, 235, 237, 239, 240, 241, 242, 261, 311, 312, 313, 314, 315, 326, 331, 393, 395, 397
  Justice Brennan, 397
  Justice Marshall, 394
  Justice Powell, 394
  Justice Rehnquist, 394
  Justice Stevens, 393
Sweden, 68, 73, 92, 93, 102
Swedish Medical Birth Register, 92, 93
syphilis, 346, 355

Taylor Diving & Salvage Co. v. *U.S. Department of Labor*, 391, 392
Tay Sachs disease, 147, 154
TCDD (see dioxin)
TCP (2,4,5-trichlorophenol), 77
teratogens/teratogenesis, 6, 55-57, 60, 62, 73-75, 204, 207, 217, 218, 221, 265, 275, 322, 351, 357, 400, 404
  teratogenicity testing, 171
tetracyclines, 356
tetraethyl lead, 69
Texas, 206
Texas Independent Ginners Association v. *Marshall*, 394, 395
textile work, 93
thalidomide, 6, 31, 55, 56, 169, 356
thiotepa, 256, 269
thiram, 269
thorium, 224
Three Mile Island Nuclear Power Plant, 32
thyroid suppressants, 356
Title VII of the Civil Rights Act (see legislation)
tobacco (see smoking)
TOK (see nitrophen)
toluene, 81, 82
tort liability, 13, 39, 279, 301, 326
  tort remedy (right to pursue), 25
  tort system, 26
  product liability, 304-307
    design defect, 306
    failure to provide, 306
    manufacturing defect, 306
toxicology studies, 8, 55, 167-170, 205, 245, 252
  general considerations, 168
  dose-response considerations, 170
Toxics Information Series, EPA, 173
Toxic Substances Control Act (TSCA) (see legislation)
toxoplasma gondii, 355
toxoplasmosis, 109, 111, 355
trichlorophenol, 32
trichlorethylene, 7
tricresylphosphate, 89
Tris (2,3-dibromopropyl), 402
Turner syndrome, 147, 154
2,4-D, 7, 78, 110, 269
2,4,5-T (2,4,5-trichlorophenoxyacetic acid), 77, 78, 110, 269
  use of, 77

Uganda, 395
ultrasound:
  exposure to, 7, 99, 100
  imaging of:
    fetal development, 159, 150, 155
    ovarian activity, 144
    peritoneal cavity, 146
ultraviolet radiation, 7, 94, 96, 97, 98

United Rubber Workers, 193
United Steelworkers of America, 193, 274
United Steelworkers of America v. *Marshall*, 391, 394, 397
uranium, 175, 224, 351

valproic acid, 164
varicella (chicken pox), 109, 111, 355
vibration, 69, 94, 104, 110
video display terminals (VDTs), 7, 100, 101, 175
Vietnam War, 78
vinblastine, 269
vinyl acetate, 89, 90
vinyl bromide, 89
vinyl chloride, 7, 88, 89, 90, 92, 170, 185, 197, 269
  standard, 185
vinyl fluoride, 89, 90
vinyl halides, 89, 90, 110
vinylidene chloride, 89, 90
vinylidene fluoride, 89, 90
Virginia, 351
visible light, 97
vitamin D, 356

Wales, 102
Walter Reed Army Medical Center, 269-274
Warfarin, 170, 269, 356
Western Electric Co., 255
West Virginia, 206, 351, 396
Whorton, Donald, 199
Willow Island, West Virginia, 251, 255, 256, 257, 259, 260
Wisconsin, 206
women workers:
  historical perspective, 34
  history of protective legislation, 235-237
  married, with children, 34, 35
  risks to, 330
Workers' Compensation, 4, 12, 13, 17, 24-26, 38, 39, 279, 298, 301, 306, 316
  exclusivity of remedy doctrine, 13, 280, 294, 295
    dual capacity exception to, 14, 295
    intentional tort exception, 14, 295
  job disability, 289-291, 297
  job relatedness (causation), 291, 293, 297
World War II, 356
Wright v. *Olin Corp.*, 249, 250, 251

X-rays, 7, 36, 37, 94-97, 100, 223, 245, 247, 248, 356, 390
  diagnostic use in obstetrics, 150
  occupational exposure, 223
xylene, 81, 82

Yusho (rice oil disease), 80

zinc, 73, 93, 188, 356
Zuniga v. *Kleberg County Hospital*, 248

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